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THE SURGICAL CORRECTION OF CALCIFIC AORTIC STENOSIS IN ADULTS

Results in the First 100 Consecutive Transaortic Valvuloplasties

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INTRODUCTION

THE EVOLUTION of the surgical correction of aortic stenosis from the unlikely adventure of Tuffier[1] to the present day has been documented. We can refer to the work of only a few individuals who, with their associates, have contributed substantially. The *life cycle* of the disease has been studied by Levine,[2] Ellis,[3] White,[4] and Nadas.[5] *The pathologic patterns* have been reviewed in standard textbooks and by Edwards[6] and McMillan.[7] Techniques of surgical access have been improvised by Glenn[8] and Swann, W. K.[9, 10] The transventricular valvulotome operations have been tried widely and with varying success by Brock,[11] Bailey,[12] Glover,[13] Muller,[14] and Merendino.[15] Open operations under hypothermia have been used by Lewis,[16] Swan, H.,[17] and Morrow.[18] Other procedures using cardiac bypass have been described by Lillehei and Varco,[19] Cooley,[20] Clowes,[21] and Effler.[33] Closed transaortic

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techniques with digital control have been advocated by Bailey,[23] Swann,[9] and by us.[24-26] Many are currently investigating prosthetic valve replacement as exemplified by the work of Sarnoff,[27] Hufnagel,[28] and Bush.[29]

What one cannot read in these discussions is the discouraging experience, largely unpublished, and available only through personal communications. This has led some cardiologists and surgeons to one of a variety of reactions: first, to conclude that the pathologic nature of aortic stenosis precludes a reasonable surgical attack until valve replacement is possible; second, that patients should be operated in an earlier phase of the disease or even before there is calcification of the cusps (this ignores the fact that many patients are not even known to have heart disease until after calcification occurs or that many patients with calcific aortic stenosis never become symptomatic); third, that surgeons should reject the patient in failure or those in the late phases of the life cycle of the disease. This interest in improving the statistical results of surgery is cold comfort to the physician already alarmed by a disease often suddenly fatal. He may be obliged to choose, with too little counsel, between the anonymity of the error of omission in "following" patients and the indictment of participating in commission should the patient die at surgery.

This complicated problem has been compounded by grouping all auscultatory evidence of obstructions in the aortic valve together, be they congenital, rheumatic or arteriosclerotic, calcific or noncalcific, young or old, compensated or in failure. In this kaleidoscope of variations in the disease clear definitions are needed in order to understand what facet is being discussed or treated.

PATHOLOGIC PATTERNS AND SURGICAL TECHNIQUES

In order to understand aortic stenosis the surgeon must study pathologic patterns and evaluate surgical techniques in the autopsy room. The "memory" provided by colored motion pictures is invaluable and in dramatic contrast to a study of formalinized specimens. We have extended this method by using the McMillan pulsator for observing normal and abnormal valves under life-like hydraulic conditions. Furthermore, the pulsator affords an opportunity to practice various surgical techniques and to evaluate the degree of restoration. Such observations combined with our clinical experience with transventricular operations led us to abandon the blind valvulotome procedure in 1954.

While our dissatisfaction with the transventricular operation grew, it was observed in autopsy specimens that calcific aortic stenosis was often most effectively mobilized by digital maneuvers, not necessarily controlled by direct vision. This experience and that of Bailey[22, 23] and Swann[9, 10] led to the development of this transaortic operation. Conversely, when the valve was flexible, elastic and noncalcific, finger dissection was often unsatisfactory and manipulation under direct vision was helpful.

The standard pathologic patterns of calcific aortic stenosis are demonstrated in Fig. 1.

SELECTION OF THE PATIENT

The selection of the patients in this series for operation has been previously considered.[25, 26] The indications appear in Table I. In addition to the usual triad of angina, syncope, or left ventricular failure, auricular fibrillation is considered an ominous finding because of the known rapid deterioration of patients with aortic stenosis once this inefficient rhythm becomes established. The diagnosis of aortic stenosis is established by the presence of a diamond-shaped aortic murmur, calcification of the aortic valve on fluoroscopy, and left ventricular hypertrophy by electrocardiogram. We consider frequent

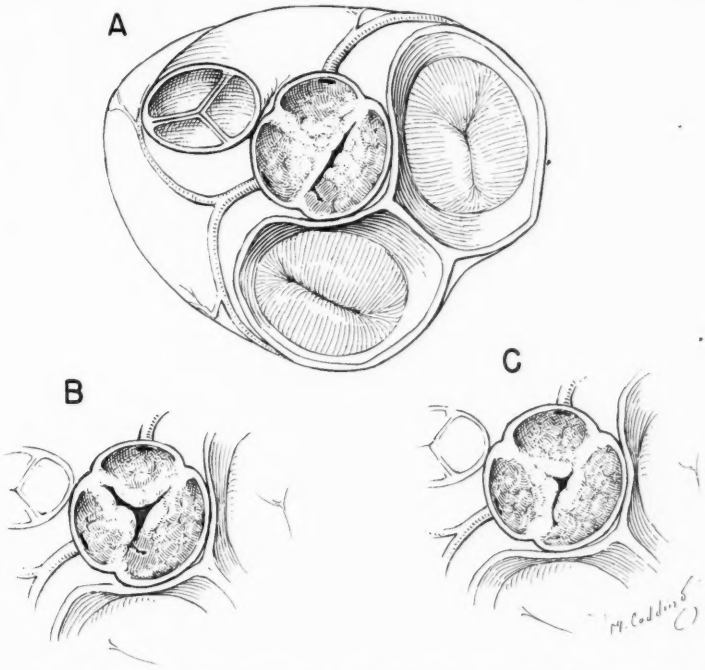

Fig. 1.—Common pathologic patterns of calcific aortic stenosis in adults. *A*, Bicuspid—occurs in approximately 40 per cent. *B*, Tricuspid—40 per cent. *C*, Punctate—10 per cent. Remainder are combinations of these types.

episodes of angina or syncope to be serious clinical manifestations of this disease. Left ventricular failure, even as manifested by dyspnea or auricular fibrillation, is an even more ominous finding. According to Ellis, in the presence of failure or fibrillation, the onset of heart pain or syncope is usually a terminal phenomenon.

In a series of 54 patients advised to have operation on similar indications but who did not accept or come to operation, 49 were dead within 6 months. It is against this background and an understanding of the technical problems presented by the calcified valve in the adult that these results must be judged.

Age has not been a deterrent to operation, since 38 of these patients were between 51 and 66 years of age. The majority (72) were males in contrast to the findings in patients with mitral stenosis.

Calcification of the aortic valve was present in 70 of the 71 males and in 20 of the 29 females. The one male without calcification (No. 75, M. S.) was found at operation to have dominant mitral insufficiency. Aortic stenosis, although present, was minimal. Hence, this experience suggests that hemodynamically significant aortic stenosis in males rarely occurs in the absence of aortic valve calcification. Of the 9 women without calcification, only one (No. 81, G. D.) had isolated aortic disease. The remaining 8 had varying degrees of associated mitral disease.

We have not eliminated any patient because of advanced congestive failure. Others had manifested significant and progressive dyspnea (Table I).

TABLE I. TRANSAORTIC VALVULOPLASTIES (100 CASES)*

PREOPERATIVE STATUS	PERCENTAGE OF PATIENTS	OPERATIVE DEATHS	LATE DEATHS	UNIMPROVED	FOLLOW-UP LESS THAN 6 MONTHS	IMPROVEMENT			
						SLIGHTLY	MODERATE	MARKED	TOTAL
Dyspnea (1)—mild to moderately severe, little or no angina or syncope	37	2	1	2	15	1	6	10	17
Dyspnea (2)—moderate to severe angina but little or no syncope	22	5	3	1	0	0	3	10	13
Dyspnea (3)—usually severe with angina and syncope	5	1	0	0	3	0	0	1	1
Congestive heart failure—admission or recent past	24	7	5	3	3	1	4	1	6
Angina—little or no dyspnea or syncope	6	0	1	1	1	0	1	2	3
Syncope—little or no dyspnea or angina	6	1	2	0	0	0	2	1	3
Totals	100%	16	12	7	22	2	16	25	43

*Preoperative and postoperative clinical status following aortic valvuloplasty as determined by Dr. Laurence B. Ellis (supported by National Heart Institute, Grant H-442).

SELECTION OF THE OPERATION

The general clinical evaluation of these patients is discussed elsewhere.[25, 26] Ellis and co-workers[3] have given additional specificity through their retrospective study of the significance of angina, syncope, failure, and fibrillation. Their emphasis on the importance of failure, even in the form of dyspnea, and of the ominous significance of auricular fibrillation are important

additions to the thinking in this field. Progressive dyspnea, in spite of medical treatment, has come to be recognized as an urgent warning that the patient is entering a late phase of the disease.

An important adjunct to the understanding of the pathologic patterns and varied clinical phases has been the observation of the patient at fluoroscopy. By this technique it is possible to make some estimate of left ventricular size and activity, of aortic arch size and activity, and thereby a differentiation between stenosis and regurgitation. Calcification is best identified by this technique and usually a differentiation of bicuspid, tricuspid, or punctate forms can be established, as well as the mobility of specific cusps. The increasing availability of image intensifiers should increase the importance of this preoperative assessment.

In the absence of a suitable prosthesis for valve replacement a choice must be made on the basis of available experience and autopsy examination of the best alternative operation for various types of aortic stenosis. We have not had the personal experience with noncalcific congenital aortic stenosis in children which would permit the selection of the optimal technique. Transventricular dilatation, however, would not seem to be definitive, and the aorta is too small for the application of an operating tunnel and for digital manipulation. Here, just as in congenital pulmonic stenosis, direct vision valvuloplasty seems logical. The size of the aorta and texture of the valve both favor open operation.

Our strong preference for the transaortic operation for aortic stenosis is based on the unsatisfactory results that followed our initial efforts with the transventricular approach. Of 23 patients operated on by this route 10 or 43 per cent were listed as operative mortalities and 10 have subsequently died. The concept that "selective insufficiency" could be borne by the hypertrophied left ventricle is now known to be erroneous. In 9 recent patients, with combined aortic and mitral stenosis, we were lured back to the simpler transventricular procedure combined with mitral valvuloplasty by the availability of improved aortic dilators, the encouraging reports of Brock, Glover, and Muller, and the limitations of the transaortic operation. The procedure, although simple to perform, has again produced discouraging results. There were four operative deaths and one late death. The inability to assess directly what is being done to the valve renders this a poor approach. It is also probable that operative aortic regurgitation is more likely to occur. This small additional experience further substantiates our conviction that the transaortic route is vastly superior and we now find little place for transventricular procedures.

Following these unsatisfactory experiences, we turned to the transaortic approach with the important addition of an operating tunnel of Ivalon* designed by Dr. Hugh E. Wilson, a former associate. This series consists of 100 adult patients with severe aortic stenosis, heavily calcified in 90. These patients suffered various degrees of congestive failure (Table I). It is hoped that a comparison of the results of this study with that of others employing other techniques in *similar patients* will indicate the most reliable procedure.

*Clay-Adams, Inc., New York 10, N. Y.

In dealing with combined mitral and aortic stenosis, the sternal splitting incision allows adequate approach to both valves. Nineteen patients in the present series have had the mitral valve explored at transaortic valvuloplasty. In 14, significant stenosis was corrected in this valve as well. The overall operative mortality rate for this combined operation compares favorably with that for the aortic procedure alone (10.5 and 17.3 per cent respectively). It is interesting to note that combined aortic and mitral stenosis was present in 53.5 per cent of the women in this series as contrasted with only 9.7 per cent of the men.

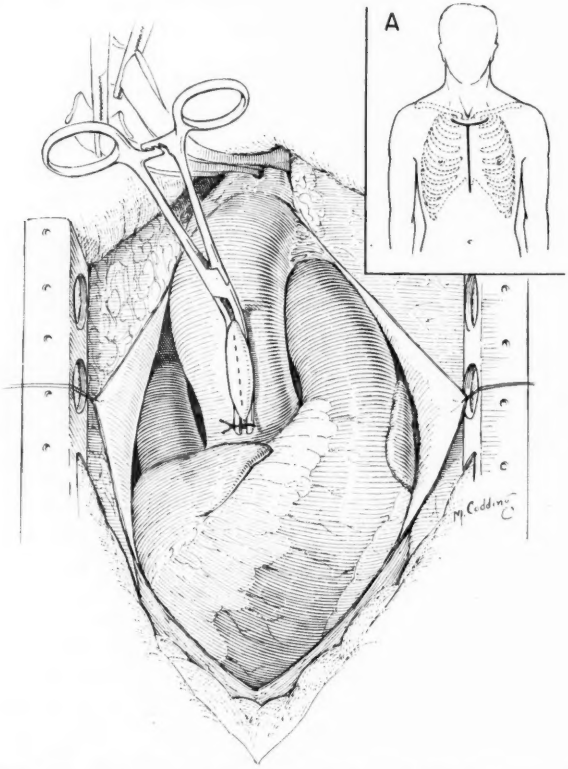

Fig. 2.—Technique of transaortic valvuloplasty—incision and exposure. *Insert*—Mid-line sternotomy. Curved Atraugrip clamp excludes a segment of ascending aorta. Rubber dam drains about right innominate and left common carotid arteries.

OPERATIVE TECHNIQUE

The operation employed is a transaortic digital manipulation of the valve employing an Ivalon sponge operating tunnel, as described and illustrated in detail elsewhere.[26]

The sternal splitting incision is important. It is well tolerated by seriously ill patients since they are operated upon in the dorsal decubitus position, allowing the pleural spaces to remain intact with both lungs available for ventilation

(Fig. 2). In addition, the placement of the aortotomy site on the anterior and most accessible portion of the ascending aorta permits support of the suture line by the reapproximated sternum. In none of these was a postoperative aneurysm observed, in sharp contrast to the 2 in whom a right lateral approach was used. One of the latter died from an aneurysm at the aortotomy site, and, in the other, limited exposure resulted in an unsatisfactory manipulation.

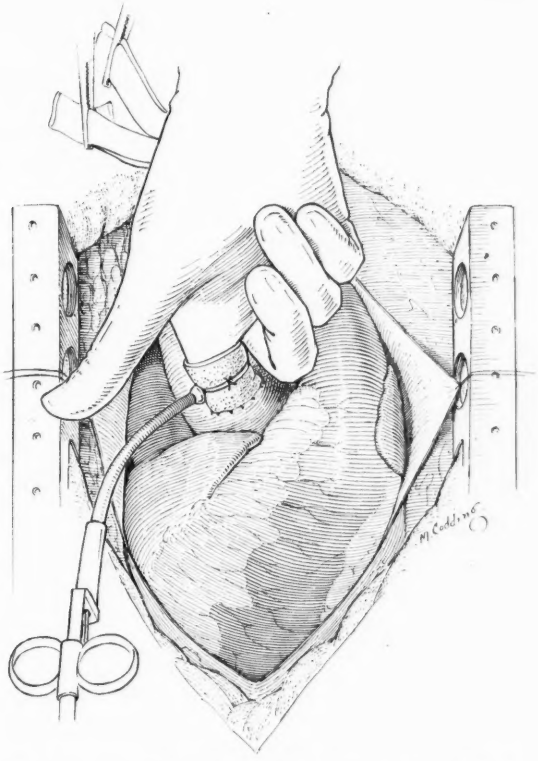

Fig. 3.—Technique of transaortic valvuloplasty. Ivalon tunnel sutured to aortotomy. Operator's finger in aorta above valve.

The Ivalon tunnel is an essential feature of this operation. In the first 28 procedures, the operative as well as late results were unfavorably influenced by the use of other materials in making the tunnels (Fig. 3). Rubber, pericardial, plastic, and cloth tunnels seem much less attractive. The sponge tunnel can be sewed in place easily and with excellent hemostasis. The maneuvers need not be hurried, as they invariably are when an alarming amount of blood loss occurs at the tunnel anastomosis. The patient's blood pressure and coronary perfusion can recover between manipulations. In this series, intra-aortic manipulation has varied from 10 to 45 minutes. There are conspicuous limitations to any technique that attempts to convert these calcific masses into effective flutter valves with maximal reduction of resistance to

forward flow and minimal production of regurgitation. The ability to assess deliberately leaflet activation by the ventricle is a striking advantage of this technique over transventricular and open operations. Another advantage of this tunnel exploration is the facility with which the stenotic orifice may be located. In the extensively calcific valve of punctate form there may be considerable difficulty in locating the orifice under direct vision when there is little flow. The visibility is only relative and any pit containing blood looks like an orifice. Contrariwise, palpation of the jet issuing from the orifice during tunnel exploration permits prompt location.

More effective mobilization of leaflets has been possible since we have learned to insinuate the fingernail, then the bare finger under the broadest zone of fusion at a hinge zone. The finger can then find the opposing fused commissure and fracture from the smooth underside by lifting upward.

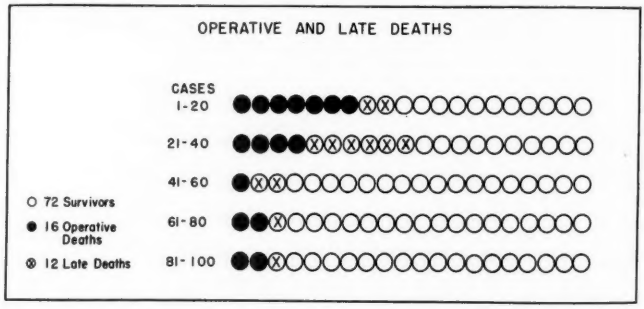

Fig. 4.—Operative and late deaths. Note reduction of operative mortality to 5 patients in last 60.

Once a fracture has started, gentle manipulation widens and stretches the commissure at the annulus until maximal mobilization of the cusps has been provided. Palpation will reveal the degree of mobilization that the left ventricle can effect in the cusps. It may be of no value to force the finger through an orifice and then assume that the effective valve area has become finger size. The ventricle may be totally incapable of producing a similar orifice. With mobilization, the leaflets may close more effectively as well as open more readily, thus reducing regurgitation as well as stenosis. Overcorrection, the *bête noire* of the field, seems more of a hazard in open surgery.

This closed technique also permits accurate timing of head vessel occlusion to prevent embolization. The first operation in this series resulted in the death of the patient from multiple calcific emboli. Head vessel occlusion for 10 to 20 seconds after each manipulation, when particles might be dislodged, has eliminated the problem of embolization.

OPERATIVE DEATHS

There were 16 deaths in this series of 100 transaortic operations. The operative mortality in the early portion of the series was high (7 of the first 20) (Fig. 4). In the last 60 patients there have been but 5 deaths (8 per cent).

Operative mortality figures afford only the crudest basis for judging a procedure but the current mortality rate of 8 per cent compares most favorably with that of the open procedures. There has been no significant difference in the operative mortality rates between the two sexes.

Four deaths were due to the production of aortic insufficiency. Two others occurred in patients with preoperative aortic insufficiency and severe congestive failure. Technical faults were responsible for three deaths (calcific emboli, postoperative hemorrhage, and tear of the base of the aorta). One other patient died on the table from cardiac arrest.

Acute arrhythmias leading to ventricular fibrillation at 12 hours, 24 hours, and 3 weeks after operation occurred in 3 additional patients. The remaining 3 deaths were not directly related to the surgical procedure (aspiration of vomitus, liver failure secondary to congestive failure, and hemorrhage from a peptic ulcer) but they do reflect the advanced nature of the disease.

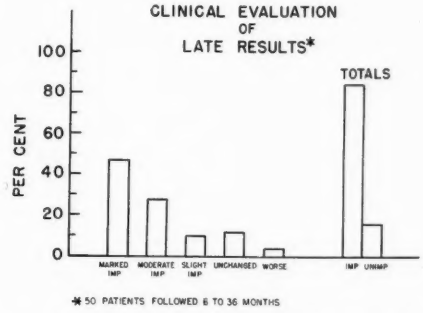

Fig. 5.—Percentage of improvement following operation in 50 patients followed more than 6 months.

FOLLOW-UP STATUS

Among the 100 patients in this series, 84 survived the operation. Late death has occurred in 12 from 1 to 31 months after surgery (average 11.9 months). Congestive heart failure was the predominant cause in 6 of these, probably due to inadequate relief of the stenosis. In 2 others the congestive heart failure was associated with aortic insufficiency. The remaining deaths were attributed to: an infected aneurysm at the aortotomy site, arrhythmia, subacute bacterial endocarditis in a patient who refused to take prophylactic penicillin, and a fresh myocardial infarction in a patient who had had his initial infarction 1 year before surgery.

Of the 72 survivors, 50 have been followed 6 months or more and 43 or 86 per cent of these are improved (Fig. 5). The majority are asymptomatic and working. These patients are being reassessed at 6-month intervals by Ellis and will be reported independently. At the time of publication the status according to clinical groups is shown in Table I. It will be seen that 88 per cent of these patients had been in overt congestive failure or had manifested dyspnea before operation.

The percentage of improvement is quite similar in all groups except those who had been in recent overt congestive failure, but even among these there was significant salvage.

HEMODYNAMIC STUDIES

Clinical follow-up studies following any operation are of primary importance, since the objective is the relief of symptoms and prolongation of life. All survivors are being followed clinically. In addition, objective hemodynamic studies before and after operation add greatly to our understanding of the procedure. We have been able to study 22 patients in this series by left heart catheterization both before and after transaortic valvuloplasty.*

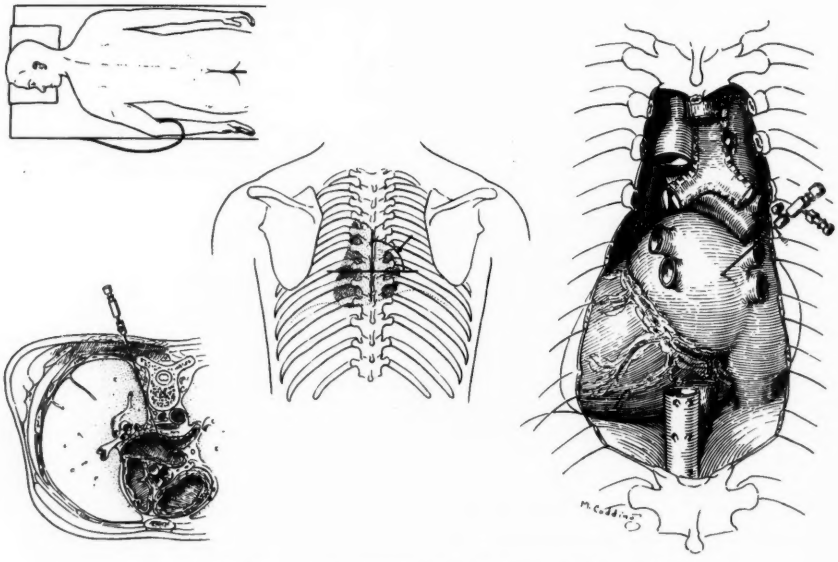

Fig. 6.—Technique of percutaneous left heart catheterization.

The technique employed in left heart catheterization has been derived from that of Fisher,[34] inserting a No. 16 thin-walled needle percutaneously into the left auricle under fluoroscopic control (Fig. 6). Through this a polyethylene catheter (PE 60) is inserted and pressures recorded in left auricle, left ventricle, and brachial artery. Cardiac output was determined by indicator dilution curves sampled at the brachial artery following left auricular injection. These curves have also been analyzed by a modification of the Korner-Shillingford formula for regurgitation and, in each case, a decision made on clinical grounds whether to consider the regurgitation to be located in the mitral or aortic area. When significant aortic insufficiency was present the

*We are indebted to Dr. Lewis Dexter, Dr. Richard Gorlin, and Dr. Walter H. Abelmann under whose auspices these catheterizations have been performed.

calculations of aortic valve area and left ventricular work have included a correction for this factor. It is recognized that the assumptions involved in the use of indicator dilution curves for the determination of regurgitation are open to some question, but at the moment they are the only method available. Nevertheless, our experience in evaluating pure mitral valvular disease would indicate that the method is usually reliable.

In the first 75 patients in the series the studies were limited primarily to diagnostic problems. When it became apparent that good clinical results were being experienced in patients in whom the catheterization data appeared less dramatic, we elected to catheterize all possible patients in the last 25 of the 100. These consecutive studies should provide accurate evaluation of the quality of our current surgery. Only 11 of the last 25 patients were studied because of other obligations in the laboratory, administrative problems, and

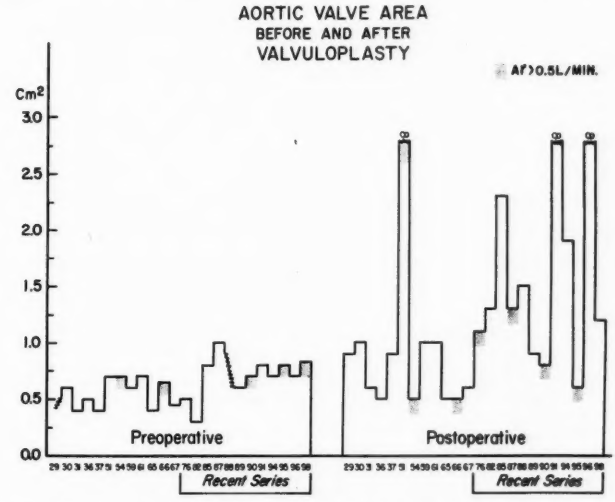

Fig. 7.—Aortic valve area calculated by Gorlin formula before and after operation.

the fact that some patients had had adequate studies elsewhere prior to reference to us. This latter series was operated on at a time when the lessons learned earlier could be applied, therefore these patients probably had a more effective operation. This can be detected in the last 11 patients represented in Figs. 7, 8, and 9.

Aortic Valve Area.—The valve area was calculated according to the hydraulic formula of Gorlin,[32] taking into account the degree of aortic regurgitation as indicated above. All but one were below 0.8 cm.² preoperatively (Fig. 7). Those with significant regurgitation are indicated by shading, since this factor produces a critical hemodynamic situation at a larger valve area than is true when stenosis exists alone. Gorlin has estimated that dynamically significant stenosis occurs at about 0.5 cm.² with pure aortic stenosis, and as high as 1.5 cm.² if associated with significant regurgitation (Fig. 10).

Postoperatively, all but 8 of the patients had valves calculated to be 0.8 cm.^{2} or more and 13 were 1.0 cm.^{2} or larger. In 3 cases, the aortic gradient was entirely eliminated indicating an effective valve area of 3.0 cm.^{2} or greater.

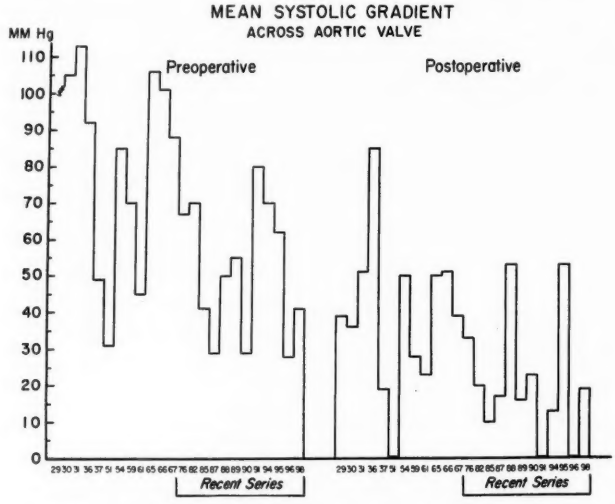

Fig. 8.—Mean systolic pressure gradient across aortic valve before and after operation.

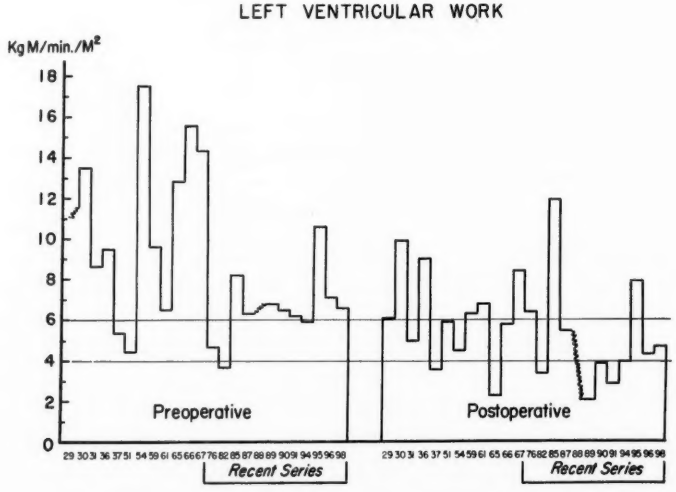

Fig. 9.—Left ventricular work before and after operation. Shaded bars indicate presence of aortic insufficiency in excess of 0.5 liters per minute.

Aortic Valve Gradient.—The preoperative mean systolic gradient across the aortic valve was greater than 40 mm. Hg in all but 4 of the patients studied and ranged as high as 113. This gradient fell postoperatively in every case but

1. In 10 patients it was reduced below 20 mm. Hg, a level now considered to be of little hemodynamic significance. The aortic valve gradient per se, without concomitant flow studies, is of limited value. Gradients in patients with low flow in failure are deceiving. This accounts for the low values recorded here (Fig. 8).

Left Ventricular Work.—Although the computed figures for valve area postoperatively were somewhat disappointing in some cases, this finding has not by any means precluded a satisfactory clinical result. The work performed by the left ventricle is perhaps of greater significance. A reduction of this factor below the critical level may reflect more accurately the relief of symptoms than does the absolute valve size. After operation, 16 of the 23 patients studied had the work of the left ventricle reduced to 6 Kg. M./min./M.^{2} or below, a level considered to be within normal range (Fig. 9).

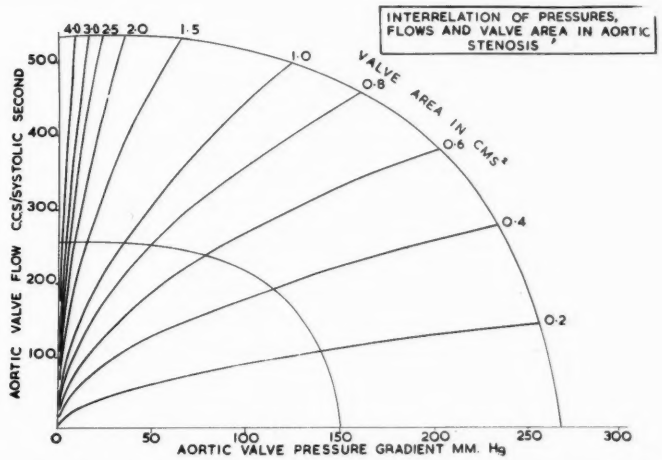
INTERRELATION OF PRESSURES, FLOWS AND VALVE AREA IN AORTIC STENOSIS
40 30 25 20 1·5 1·0 0·8 0·6 0·4 0·2
VALVE AREA IN CMS.²
AORTIC VALVE FLOW CCS/SYSTOLIC SECOND
500 400 300 200 100
0 50 100 150 200 250 300
AORTIC VALVE PRESSURE GRADIENT MM. Hg

Fig. 10.—Diagram showing relation of pressure to flow for any given degree of aortic stenosis. Note minimal increment in flow despite marked increase in gradient in valves of 0.5 cm.^{2} area or less. (Gorlin et al.: Am. J. Med. 18: 855-870, 1955.)

DISCUSSION

A review of this series of 100 patients who have had a transaortic valvuloplasty for the relief of aortic stenosis emphasizes several points. It is clear that the symptomatic patient with aortic valvular obstruction has a disease that is often fatal within a matter of months as documented by our control series. As Wood[31] has pointed out, the prognosis for relief or survival on medical therapy alone is poor. The operation presented, although not curative in the majority, has offered good palliation to many at an acceptable operative risk. We cannot agree with those who urge early operation "before calcification occurs" simply to minimize the technical problems. Nevertheless the development of symptoms in a patient with the murmur of stenosis should warn the physician of impending rapid deterioration and prompt consideration of surgical relief is in order. Left heart catheterization may clarify the problem.

At the other end of the spectrum is the patient in congestive heart failure who should not be denied operation simply because of increased operative risk. He has a malignant disease on medical treatment yet surgery may offer some salvage.

When hemodynamic evaluation can in some instances show so little change yet the clinical improvement be great, or vice versa, we realize that even these techniques leave something to be desired. Gorlin[30] has recently been studying coronary flow in these patients. Patients with aortic stenosis show a fixed coronary flow at rest, on exercise and with nitroglycerine. Presumably, the excessive ventricular work calls for maximal coronary dilatation. It will be significant if further studies show that the fixed coronary flow of advanced aortic stenosis can become variable after surgical correction. Such a study might help us in the selection of patients for operation and also facilitate the evaluation of results.

SUMMARY

1. One hundred patients with symptomatic aortic stenosis have had surgical correction by a transaortic procedure. There have been five operative deaths in the last 60 patients (8 per cent). Twelve late deaths have occurred in the entire series.

2. Clinical appraisal of the survivors, followed from 6 to 36 months, indicates that 86 per cent are improved.

3. Hemodynamic evaluation by left heart catheterization before and after operation shows that the valve area is significantly increased, the mean systolic gradient reduced, and that the left ventricular work falls.

4. Serious consideration for surgical relief is advised in all patients with symptomatic aortic stenosis.

5. A plea is made for careful preoperative and postoperative clinical and hemodynamic evaluation of patients with aortic obstruction. This should facilitate the proper choice of closed or open operation in the various patterns of disease.

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DISCUSSION

DR. ELMER P. R. MAURER, Cincinnati, Ohio.—I would like to emphasize the importance of an observation made by Dr. Harken that has to do with the preoperative evaluation and determination of the etiological background in choosing the method of surgical approach for aortic stenosis. The results obtained will depend in great part on this determination. In aortic stenosis resulting from rheumatic fever, one would anticipate commissural fusion characteristic of this disease and the transaortic approach might very well be anticipated to give good results. In the arteriosclerotic, calcified form of aortic stenosis, no commissural fusion will be anticipated and I think the percentage of good results will be decreased.

(slide) With either the open or closed approach, it is hopeless to expect good results in those situations where one has complete replacement of the aortic valve by a calcified block. Taking into consideration all aspects of the problem, including the added risk of open aortotomy with electric cardioplegia or retrograde coronary perfusion with extracorporeal circulation, we have preferred to use a transsternal plastic tunnel closed approach in those individuals who have a definite rheumatic history and the classical commissural fusion.

(slide) The valve is approached through the tunnel and commissurotomy is completed by finger fracture or cutting technique.

(slide) This shows the aorta closed.

We have encountered no problems in postoperative hemorrhage utilizing this approach.

We certainly believe that open approaches are mandatory in all instances of congenital aortic stenosis. I doubt very much whether any approach is satisfactory in those instances where the aortic ring and valve is completely replaced by calcium. Perhaps in the future we may find ourselves exploring the aortic valve by one of the closed techniques, being prepared to proceed with the open operation if it seems impossible to do it by the closed approach.

In summary, we have preferred transaortic commissurotomy, utilizing the plastic tunnel developed by Dr. William K. Swann, in those instances where a definite history of rheumatic fever is established, the stenosis is of short duration, and no calcium is seen on fluoroscopic examination of the aortic valve. All other stenotic aortic valve lesions are operated on by the open approach.

DR. ROBERT P. GLOVER, Philadelphia, Pa.—Dr. Harken has presented his subject in his usual masterful way. Whereas we agree wholeheartedly that, at the present moment, closed methods for the surgical treatment of acquired aortic stenosis are preferable, it is our feeling that the transventricular route to the aortic valve is simpler, faster, and safer than the aortic approach and will yield a more satisfactory functional clinical result in a greater number of patients.

(slide) From 1951 to the present we have used transventricular commissurotomy for aortic stenosis in 87 consecutive cases. During this period, twice this number have either refused surgery or have been turned down and each of these has long since died. There has been no inclination to employ other techniques, for acquired calcific stenosis presents a derangement in anatomy and pathology incompatible with definitive, restorative reconstruction in the ordinary sense of the term. In the majority of such cases, the valve is indurated and calcified to the point of complete replacement and distortion of normal valvular tissues. It is an immobile "concrete dam." Under these circumstances and short of *total resection* of this scar with suitable prosthetic replacement, there is no advantage to employ techniques (complicated, tedious, time-consuming, and of considerable risk) which provide for either direct palpation or visualization of the valve. To be sure, ultimately open surgery with valvular replacement will be the effective treatment, but until such a valve replacement becomes available it must be recognized that safe and effective palliation is all that can be offered. This can best be accomplished through the left ventricle.

Originally, most surgeons used the transventricular approach with uniformly poor results. Large, bulky dilators requiring constricting purse-string sutures on the left ventricular myocardium resulted in stellate lacerations of this muscle with its attendant hemorrhage and arrhythmias and were responsible for the high mortality. Our first 37 consecutive cases were no exception for, within the first month of surgery, the mortality was 46 per cent, obviously intolerable. To be sure, these deaths were in patients already in advanced left ventricular failure, and poor risks. Surprisingly, however, 11 or 30 per cent of this group are still living and improved 3 to 7 years after surgery, indicating that, when tolerated, split-dilatation of these valves provides reasonable palliation.

(slide) Results such as these indicated the left ventricular approach in the minds of most, hence the search for and employment of other methods. Our technique through the left ventricle was changed. By using the Brock two-bladed dilator (no larger than a lead pencil) small, one-centimeter incisions in the myocardium only were necessary and no purse-string or stay sutures were needed. Manipulation and trauma to the left ventricle have been minimal and blood loss rarely exceeds 2 to 3 ounces. The improvement in results has been dramatic. Since 1954, in 50 consecutive cases, there has been an operative mortality of 6 per cent (3 cases). Of the surviving patients, 70 per cent have been greatly improved and are remaining so (1 to 3 years). Aortic insufficiency with diastolic levels below 30 mm. Hg has occurred in only 3 patients. There have been no embolic phenomena in any of the patients receiving aortic commissurotomy alone (3 cerebral emboli with one death occurred in combined aortic and mitral commissurotomies of which there were 24 in the 87 cases). Pressure gradients across the aortic valve taken in all 50 cases, pre- and postcommissurotomy at the time of surgery, have resulted in a drop from an average of 78 mm. Hg to 27 mm. Hg. This hemodynamic improvement, while not perfect, can hardly be improved upon when one remembers the pathology present the mere presence of which will, because of its immobility, provide some obstruction to left ventricular outflow whether the commissures are open or not.

Thus, we are encouraged. The fault was not in the approach through the left ventricle but in the technique employed. Better results will be obtained only when patients with this disease are referred for surgery earlier in their pathologic course or when these scarred valves (they no longer have the right to be called valves) can be totally resected and replaced by a suitable prosthesis. Until then, from the practical standpoint, palliative surgical treatment as described offers the patient his best opportunity for an improved clinical state.

DR. WILLIAM W. L. GLENN, New Haven, Conn.—Dr. Harken and his colleagues have reported a remarkable experience with a most difficult valvular lesion. We have had a similar protracted interest in the transaortic approach to the functioning aortic valve. Extensive experience with this technique, such as Dr. Harken's large series, is of the greatest importance in the successful relief of calcific aortic stenosis. In a smaller series, we have on two occasions produced uncontrollable aortic regurgitation by the combined digital and instrumental dilatation of the aortic valve in the functioning heart and we believe that, in both instances, it was due to avulsion of one aortic cusp from its point of attachment to the wall of the aorta. Further, it has never been possible for us to open more than two commissures by this method, and frequently it has been possible to open only one commissure.

Despite our continued interest in the diverticulum approach to the pulmonary valve and, when indicated, the mitral valve, we have come to the belief that for us, and perhaps for some others of lesser experience than Dr. Harken, the indications for the diverticulum approach to calcific aortic stenosis should be limited to those patients whose general condition demands that something be done due to the presence of severe symptomatology. One should bear in mind, however, that unless an unusual local situation is found, such as a predominantly fibrous valve, the valvulotomy may be less satisfactory than one might obtain with open techniques. As a case in point (slide), we have recently operated on a patient with calcific aortic stenosis and severe congestive failure. The patient lost nearly 60 pounds of fluid in the month prior to surgery. A good split of two of the commissures was obtained through a diverticulum sutured to the aorta. However, the third commissure could not be opened due to the extensive calcification of the valve. He survived operation and was apparently improved for a period of 6 months, when he suddenly died. The slide shows the extensive calcification and immobility of the valve cusp despite separation of the commissures. Finally, the ultimate result in calcific aortic stenosis may be determined by several factors—the degree of immobility of the valve leaflets; refusion of the commissures postoperatively, particularly where the leaflets are markedly calcified; and, finally, the limits of myocardial reserve.

DR. CHARLES P. BAILEY, Philadelphia, Pa.—I have enjoyed Dr. Harken's paper very much and am glad that he emphasized the commissure. However, a heavy responsibility rests upon me; first, for originally promulgating the closed approach to the stenotic aortic valve and, second, for failing to recognize the necessity for bringing about flexibility of the surgically separated valve segments. Today it seems to me that it is wrong to continue these closed techniques for the reasons Dr. Scannell showed so well yesterday. The valve structure is not simple and there is much more advantage in operating by an open technique than is the case with the mitral valve. We feel that, while many patients are clinically benefited by the closed procedures, it is only because a relatively slight enlargement of the orifice beyond the critical size brings about a rather great clinical improvement. Postoperative physiologic studies by Goldberg and others have shown that we do not accomplish much opening of the stenotic valve by any closed method. It is not surprising that many of our surviving patients are beginning to show evidences of recurrence of the stenosis and will require further (more adequate) intervention in the future. For this reason, 86 cases ago we decided to do only open operations for aortic stenosis. When we look at the valve it is not the same as feeling it digitally or palpating it instrumentally.

(slide) This is a valve which may be considered as typical or reasonably representative of these cases, although I know that there are very many variations. Note that the calcification is limited to the concave or "spongiosa" layer of the cusp. The convex or "fibrosa" layer would be relatively thin and flexible if it were dissected away from the former. By a painstaking technique, somewhat analogous to soap sculpture, it is possible to remove the extremely scarred and calcified spongiosa layer. By this means not only is a great deal of flexibility imparted to the mobilized valve cusps, but also a significant degree of "lengthening" of the cusp is produced which renders the valve more competent.

DR. DWIGHT E. HARKEN (Closing).—Thank you, gentlemen, for your discussion and your nice comments. Thank you, Dr. Bailey, for your reference to the removal of calcium as "sculpturing" reconstruction. We have been unable to remove the calcium without producing hopeless defects. Furthermore, we can assess what the ventricle can do for valve motion better with the finger during the closed operation.

Dr. Glover and Dr. Maurer have accepted my basic reasoning. I'm not quite sure that I can entirely return the compliment, for the limitations of the transventricular technique seem virtually prohibitive to us. With Dr. Glenn's position I agree, however, that the technical advantages of a properly used Ivalon tunnel seem substantial. Dr. William Glenn has been a pioneer in the use of operating tunnels for intracardiac surgery.

In closing, I cannot possibly comment adequately on all the excellent points made by the discussers. It must suffice to say that we have patients in a symptomatic phase of a highly lethal disease. We recognize certain variations in the pathologic process. It seems to us that open operation has advantages in noncalcific aortic stenosis. Open operation certainly has a preferred place in subaortic stenosis in children. Conversely, heavily calcified stenosis in adults (with adequately large aortas) has been handled in a fairly but not perfectly satisfactory manner by this Ivalon operating tunnel in a transaortic retrograde fashion.

We need to pool and compare our results when operating on similar pathologic patterns in similar phases of the life cycle of this disease in order to adopt the most appropriate operations and to make the best recommendations to others. This evaluation must include operative and late mortality, pre- and postoperative clinical evaluation and, imperfect as it is, the most exhaustive, objective hemodynamic studies before and after our intervention. Only in this way can we work responsibly until the "perfect" operations are available.

Again, thank you.

SURGICAL TREATMENT OF THIRTY-FIVE CASES OF DRAINAGE OF PULMONARY VEINS TO THE RIGHT SIDE OF THE HEART

Henry T. Bahnson, M.D., Frank C. Spencer, M.D. (by invitation), and Catherine A. Neill, M.D. (by invitation), Baltimore, Md.*

SINCE the development of methods for accurate diagnosis of cardiac defects, anomalies of the pulmonary venous return have been reported with considerable frequency. Partial anomalies of the pulmonary veins have been often encountered in the repair of atrial septal defects and various methods have been described for closing the defect and concomitantly correcting the anomalous venous return.[5, 8, 10, 15]

There have been a number of attempts to treat the totally abnormal return, all aiming to direct the pulmonary flow to the left atrium but attempting to do this by several different methods. Muller[19] first reported partial correction by anastomosis of the venous trunk from the left lung to the left atrial appendage, and complete correction of several types of totally abnormal return has since been reported.[6, 8, 11, 16-19] The purpose of this report is to describe our surgical experiences with 35 cases of anomalous pulmonary venous return, in 16 of which all the pulmonary veins drained abnormally. These represent 38 per cent of the 92 patients treated during the past 9 years for anomalous venous return or atrial septal defect, the condition which the former most resembles clinically.

EMBRYOLOGY

The current, most widely held explanation of the developmental abnormality states that the pulmonary venous plexus arises from differentiation of the splanchnic plexus around the developing lung.[1, 21] The primordial cardiac end of the pulmonary vein develops as a small endothelial pouch growing from the atrium on the left of the developing septum and extending toward the pulmonary plexus. Simultaneously, the systemic veins are developing from fusion of the anterior and posterior cardinal veins on either side to form the common cardinal vein or duct of Cuvier. Both common cardinal veins join the sinus venosus. This later is absorbed into the heart, the right common

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cardinal vein becoming the proximal portion of the superior vena cava, the right posterior cardinal vein becoming the azygos, and the right anterior cardinal vein the superior vena cava distal to the azygos. The left common cardinal vein is absorbed into the heart as the coronary sinus. More distally, the left common cardinal vein becomes the oblique vein of Marshall and the primitive posterior cardinal vein persists as the highest intercostal vein. Should the pulmonary plexus and the pulmonary vein extending out from the atrium fail to meet, these systemic veins or their remnants form ready substitutes. The connections are already present joining the splanchnic-pulmonary plexus to the cardinal system.

Neill's grouping of anomalies of the pulmonary venous system is convenient for surgical purposes. She suggests that drainage of the pulmonary veins into the *right atrium* might occur, if the atrial septum develops abnormally far to the left. Drainage into the *right superior vena cava*, azygos, or right innominate vein would represent persistence of communication between the pulmonary venous plexus and the right horn of the sinus venosus and right common cardinal vein. Drainage into a persistent *left superior vena cava*, coronary sinus, or left innominate vein might result from persistence of connections with the left common cardinal system. In such an instance, once the pulmonary venous plexus communicates with the left horn of the sinus venosus and left cardinal system, three possibilities arise. The cardinal system may atrophy proximal to the entrance of the pulmonary veins, in which case the pulmonary veins will drain into a left innominate, or a persistent left superior vena cava (or left vertical vein). If the cardinal system atrophies distal to the entrance of the pulmonary veins, the ultimate connection is to the coronary sinus. In some instances, the entire cardinal system persists and the pulmonary veins empty into a left superior vena cava connecting with the coronary sinus as well as the innominate. Neill's fourth group consists of connection with the portal vein or ductus venosus, remnants of early drainage into the umbilicovitelline system.

In all instances, except anomalous drainage into the right atrium, it is presumed that the primordial pulmonary vein does not develop in the atrium, does not establish contact with the developing pulmonary plexus, or loses this connection once made.

Edwards and associates[18] have added an additional group of patients in which the connections of the common pulmonary vein to the left atrium are abnormal, although all the pulmonary venous blood may return to the left side. In such a case, a tri-atrial heart arises with two chambers in the left supraventricular region, the upper one arising from the common pulmonary vein, the lower from the left atrium, and the septum between being the narrowed junction of pulmonary vein and atrium. This condition will not be included in the subsequent discussion.

DIAGNOSIS

When one or two of the pulmonary veins drain directly into the right atrium and the remainder normally to the left, the clinical picture may be

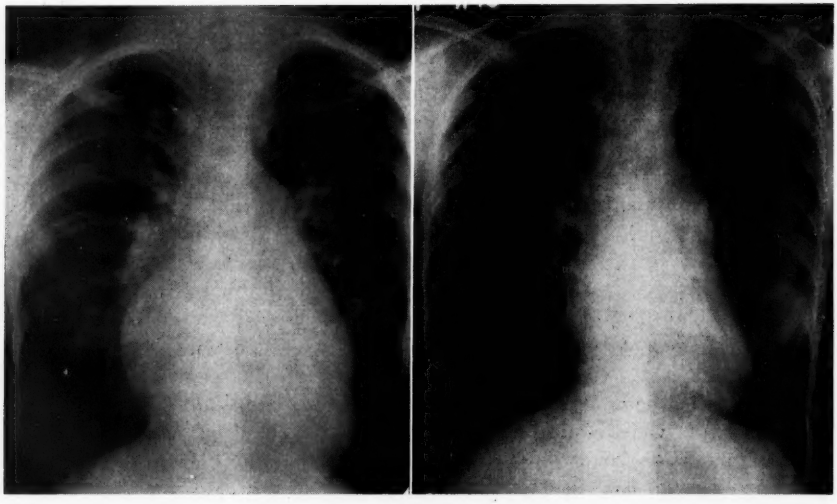
identical with that of a simple atrial septal defect. It has been stated that partial anomalous venous return is associated with higher pulmonary blood flows and pressures than those seen with simple atrial septal defect, but this has not been a consistent finding in the present series. We have noted, however, in the electrocardiogram of these patients, a more marked degree of right ventricular hypertrophy than is usually associated with a simple atrial defect with comparable pulmonary artery pressures. The correct diagnosis of the partial anomaly may be suggested by electrocardiogram, by the occasional visualization of the anomalous vein on the plain roentgenogram, by the catheterization of the vein or veins directly from the right atrial wall or superior vena cava, or by visualization with angiocardiography. Swan and associates[25] have developed methods, including the injection of indicator dyes, by which one may determine whether the drainage is anomalously to the right side of the heart. In some instances of simple atrial defect, however, the drainage from one or the other lung may be predominantly to the right. Thus, they point out a distinction between abnormal venous drainage and abnormal venous connection, the two not necessarily being the same.

In the presence of a totally abnormal pulmonary venous drainage, both systemic and pulmonary venous blood mix in the right atrium so that blood in all other cardiac chambers is of similar oxygen saturation. In some instances, due to streaming, pulmonary blood may contain a higher oxygen content than systemic arterial. Such patients must have peripheral arterial unsaturation, although with a high pulmonary flow this may be of such small degree that it is not noticed clinically without analysis of arterial blood. The total anomaly is most apt to cause symptoms and failure early in life and be associated in the absence of failure with excessive pulmonary blood flow and greater tendency to increased pulmonary vascular resistance. In Keith's series,[14] the majority of the patients died before 6 months. The size of the atrial septal defect, or foramen ovale, is probably important in the onset of failure, forming an obstruction when small. In addition, the left atrium and ventricle are usually underdeveloped.

Since anomalous drainage of the pulmonary veins into the *right atrium* complicates the operative problem only very little, it is not of great surgical importance to identify the anomalous veins prior to operation. The anomalous return in Case 1 in which all pulmonary veins entered the right atrium was strongly suspected because of the unusual disability, the large shunt into the atrium, and slight arterial unsaturation.

When the anomalous drainage is into the *superior vena cava*, the anomalous veins may be seen on a plain roentgenogram in a slightly higher than normal position (Fig. 1, *A*). This can be confirmed by angiocardiography or by cardiac catheterization when arterialization is discovered in this region, more readily demonstrable when the drainage is high in the vena cava or into the innominate. When all pulmonary veins enter the superior vena cava, this structure usually shows dilatation radiologically.

Drainage of the pulmonary veins into the *coronary sinus* may be accompanied by great cardiac enlargement and severe cardiac failure even in early



A. *B.*

Fig. 1.—Case 25. Pre- and postoperative roentgenograms (*A* and *B*, respectively). The pulmonary vascular markings enter the cardiac silhouette on the right at a slightly more rostral level than normally. Much of the cardiac enlargement is due to the right atrium.

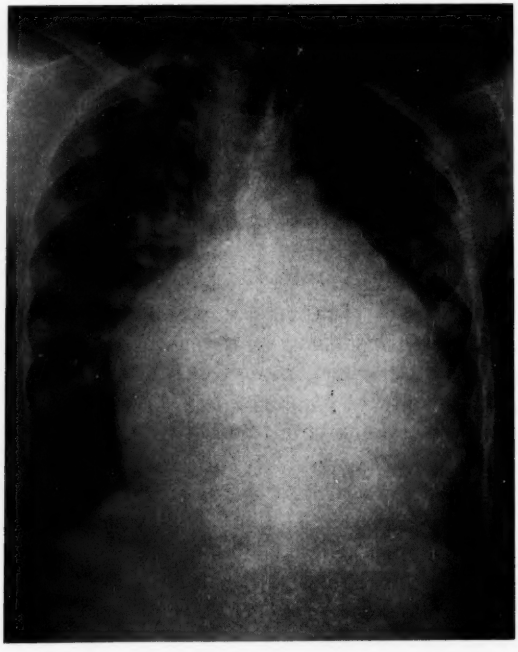
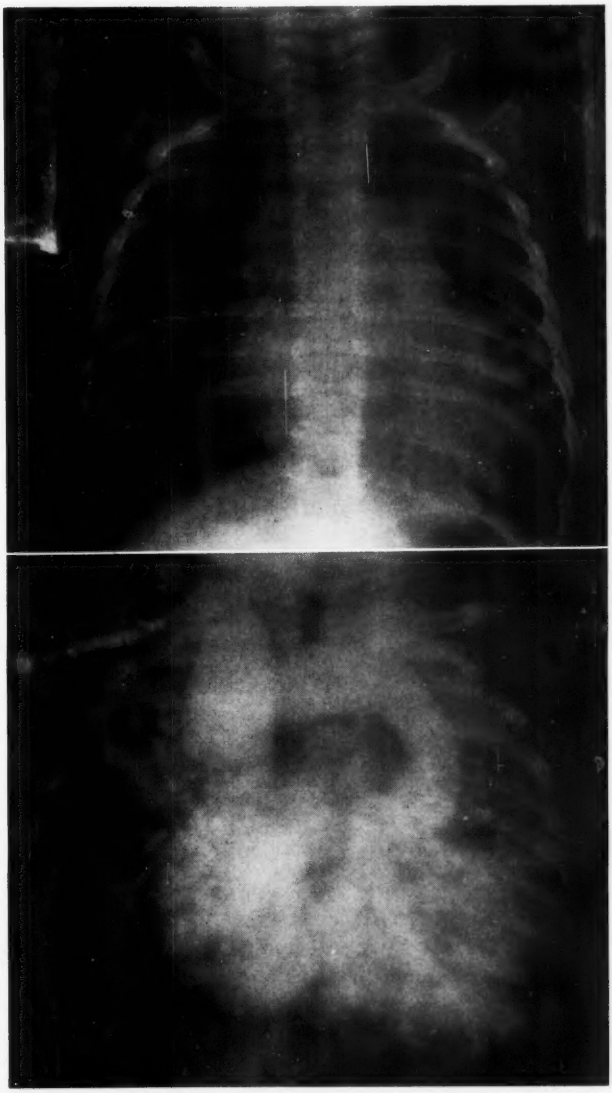


Fig. 2.—Case 28. Total pulmonary venous drainage to the coronary sinus. The boxlike shelf to the upper right border is characteristic on the roentgenogram.

A.



B.

Fig. 3.—Case 34. Total anomalous drainage into left superior vena cava in an infant. The "snowman" appearance is not readily evident on the plain roentgenogram but is clearly seen on the angiocardiogram.

childhood. The contour of the heart is unusual, the right atrium being large and boxlike with a somewhat horizontal upper border (Fig. 2). This contour when present appears characteristic but unfortunately was absent or had not yet appeared in the one infant encountered with this condition.

When all pulmonary veins drain to the *left superior vena cava*, there is a "snowman" or "figure-of-eight" appearance on the roentgenogram which has been extensively illustrated and is due to the dilated left superior vena cava (or vertical vein), innominate vein, and right superior vena cava, together forming a rounded shadow above the globular heart. This typical radiologic appearance is less easily recognized in infants (Fig. 3, *A*) but may be readily demonstrated angiocardiographically (Fig. 3, *B*) or by catheterization with the shunt demonstrable into the innominate vein.

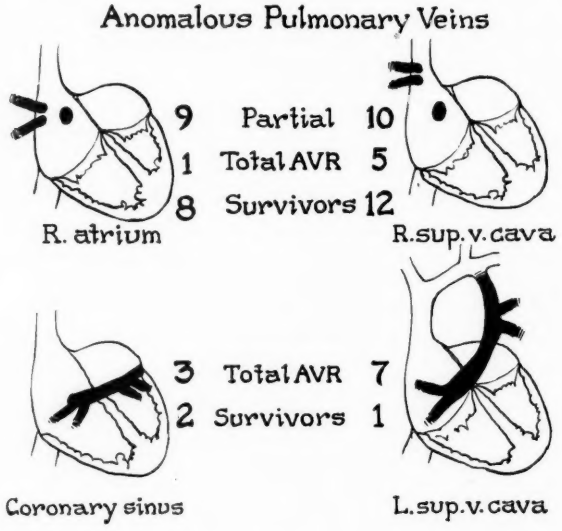


Fig. 4.

SURGICAL TREATMENT

The subsequent discussion is based upon experience as follows: There were 10 cases in which pulmonary veins entered the right atrium, in one instance both right and left sides draining anomalously. In 10 cases, some or all of the right pulmonary veins drained into the superior vena cava and in 5 all pulmonary veins from both lungs drained into a chamber which entered the right superior vena cava. All pulmonary veins drained to the coronary sinus in 3 cases and in 7 cases all pulmonary veins entered a persistent left superior vena cava (Fig. 4 and Table I).

The present method of hypothermia used with most of these cases has been reported[24] but consists of surface cooling to 30° to 32° C., mild alkalosis

TABLE I

NAME AND NO.	AGE	METHOD OF REPAIR	OTHER ANOMALIES	RESULT	REMARKS
Pulmonary Veins Entering Right Atrium					
Totally Anomalous:					
Case 1. S. H. 717950	15	Hypothermia; direct suture	None	Good	Total anomalous return to right of septal rim; septum incised to effect transposition of veins
Partially Anomalous:					
Case 2. E. B. 626696	44	Atrial well	Pulmonary thrombosis	Died	Severe pulmonary hypertension and thrombosis
Case 3. C. A. 736541	13	Hypothermia; direct suture	None	Good	Superior pulmonary vein anomalous
Case 4. L. F. 662181	5	Hypothermia; direct suture	None	Died	Unexplained severe acidosis; right inferior pulmonary vein anomalous; earlier attempt to close defect with purse string unsuccessful and sutures subsequently removed because of infection
Case 5. N. M. 768002	6	Hypothermia; direct suture	None	Good	Septum incised to effect transposition of all right pulmonary veins
Case 6. F. A. 742498	7	Hypothermia; direct suture	None	Good	Anomalous right inferior pulmonary vein only
Case 7. R. B. 622376	11	Hypothermia; direct suture	None	Good	Right superior and middle veins anomalous
Case 8. B. B. 675489	10	Hypothermia; direct suture	Left SVC	Good	All right pulmonary veins into right atrium; septum incised to transpose veins
Case 9. L. J. 760337	19	Hypothermia; direct suture	None	Good	1 cm. foramen ovale, both right pulmonary veins into right atrium; septum incised to effect closure
Case 10. P. McL.*	21	Hypothermia; direct suture	None	Good	Right superior pulmonary vein anomalous; septum incised to effect transposition of veins
Right Pulmonary Veins Into Right Superior Cava					
Case 11. J. J. 633834	6	Atrial well	None	Died	Died shortly after opening atrial well
Case 12. E. B. 735041	45	Partition of cava; purse string	None	Good	Superior and middle lobe veins anomalous
Case 13. E. B. 701657	7	Hypothermia; partition	None	Good	Superior pulmonary vein anomalous; SVC pressure 12 mm. Hg after closure

*Treated at the Royal Prince Alfred Hospital, Sydney, Australia.

Cont'd on next pages.

TABLE 1—CONT'D

NAME AND NO.	AGE	METHOD OF REPAIR	OTHER ANOMALIES	RESULT	REMARKS
Case 14. M. S. 639709	12	Hypothermia; partition	None	Good	Superior pulmonary vein anomalous; SVC pressure 12 mm. Hg after closure
Case 15. B. M. 716418	15	Hypothermia; partition	None	Good	Superior pulmonary vein anomalous; SVC pressure 26 mm. Hg after closure
Case 16. P. B. 741548	11	Hypothermia; partition	None	Good	Superior pulmonary vein anomalous
Case 17. C. W. 796284	7	Hypothermia; partition	None	Good	Superior and middle lobe veins anomalous; SVC pressure 20 mm. Hg after closure; postoperative chylothorax
Case 18. A. F.*	33	Hypothermia	Left SVC	Good	Superior and middle lobe veins anomalous; right SVC directed into septal defect under direct vision; right SVC ligated above; left SVC present
Case 19. L. H. 783712	11	Hypothermia	Left SVC	Good	Same as Case 18
Case 20. R. J. 602898	31	Hypothermia; partition	None	Good	Superior and middle lobe veins anomalous
All Pulmonary Veins Draining to Right Superior Cava					
Case 21. K. F. 506600	4	None	None	Died	Exploration on right only, in 1949; later anastomosis on left planned; right pulmonary artery coursed between common pulmonary vein and left atrium
Case 22. R. B. B 03237	6/12	Atrial well	None	Died	Anomaly not fully diagnosed until autopsy; arrest on closure of foramen ovale
Case 23. E. C.	7	Purse string; partial ligation of cava	None	Incomplete repair	Right SVC directed through septal defect to left atrium; SVC obstructed above entrance of veins
Case 24. D. D. 613723	7	Hypothermia; partition	None	Good	Right pulmonary artery coursed between left atrium and common pulmonary vein; SVC pressure rose 2 mm. Hg
Case 25. B. L. 604034	13	Hypothermia; partition	None	Good	Initial repair obstructed pulmonary veins; revised with another period of inflow occlusion
All Pulmonary Veins Draining to Coronary Sinus					
Case 26. J. R. 475646	13	Bypass	Patent ductus, tricuspid insufficiency	Good	Small foramen ovale; Septum removed between coronary sinus and left atrium, resulting defect of atrial septum sutured

TABLE 1—CONT'D

NAME AND NO.	AGE	METHOD OF REPAIR	OTHER ANOMALIES	RESULT	REMARKS
Case 27. D. H. B 38052	2	Hypothermia	None	Died	Severe cardiac failure; died after exploration with inflow occlusion
Case 28. F. A. 784086	15	Bypass	None	Good	As in Case 26; initial repair obstructed pulmonary veins, revised with additional bypass
All Pulmonary Veins Entering Left Superior Cava					
Case 29. J. G. 508089	2	Left atrial-left pulmonary vein anastomosis	None	Died	Anastomosis of left inferior pulmonary vein to atrial appendage, trunk constricted rostrally
Case 30. C. B.	4	Left atrial-left pulmonary vein anastomosis	None	Died	Anastomosis of left atrial appendage to venous trunk; constriction rostrally
Case 31. A. S. A 95474	4/12	Left atrial-left pulmonary vein anastomosis	None	Died	Anastomosis of left atrial appendage to venous trunk; constriction rostrally
Case 32 B. B. 681276	28	Left atrial-left pulmonary vein anastomosis	None	Died	Aortic graft used to join left atrium and venous trunk
Case 33. J. H. B 36459	10/12	Hypothermia	None	Died	Attempted transposition of right SVC to foramen ovale; improper suture
Case 34 N. B. B 44197	3/12	Bypass	None	Died	Small infant in severe failure; attempted transposition of right SVC
Case 35. J. D.*	20	Bypass	None	Good	Right SVC directed to left atrium through foramen ovale; azygos and right innominate veins ligated

by hyperventilation, and gravity perfusion of the coronary arteries during inflow occlusion. Two periods of inflow occlusion of 9 minutes each were used in Case 26 but, in most instances, repair was accomplished in less than 8 minutes.

The pump-oxygenator used for cardiopulmonary bypass was the stationary screen oxygenator as modified by Gaertner and Kay.

In most instances, operation has been performed through the right chest only, the fourth interspace being entered. When additional exposure is required, as for visualization of the left superior vena cava, the sternum may be transected but rarely does the left chest need to be entered for treatment of anomalies on the right side. In earlier experience, in attempting to anastomose the auricular appendage to the persistent left superior vena cava, an approach through the left side was, of course, used.

The importance of thorough dissection and visualization of all vessels in the operative field cannot be overemphasized. Although, in some cases, anomalous pulmonary veins were suspected from clinical or catheterization data, in most instances the partial anomalous drainage was first diagnosed with certainty only after such dissection. One should be certain of visualizing veins draining all areas of the exposed lung, and clearly identify, within the pericardium or at the site of entering the superior vena cava, the termination of such veins. The position of veins can then be further delineated by palpation within the heart through an incision in the wall of the atrium or the tip of the atrial appendage. In palpating within the heart a plan should be developed, our own being to examine the superior and inferior venae cavae, coronary sinus, the atrial septum and, if the septum is not intact, the origin of right and left pulmonary veins through the defect, the left atrial appendage, the mitral valve, and the tricuspid valve. Accurate examination of all these structures is not possible in infants with small hearts and it is our belief that particularly in such children open visualization is needed; this requires additional time of circulatory interruption and hence a cardiopulmonary bypass should be used.

Pulmonary Veins Entering Right Atrium (Table I).—When pulmonary veins drain anomalously into the right atrium in the region of an auricular septal defect no difficulty is usually encountered in suturing the anterior rim of the defect to the right of the pulmonary veins in the repair. In some instances, veins enter the right auricle just above or below the defect and adjacent to intact septum. In such instances, incision of the septum is required; this should be done posteriorly, adjacent to the orifice of the anomalous vein, and this flap of septum may then be sutured anterior and to the right of the pulmonary veins (Fig. 5). In one patient (Case 1), treated under hypothermia, the rim of the septal defect was unusually far to the left and all right and left pulmonary veins drained into the right atrium to the right side of this septal rim. Regardless, after incision to enlarge the defect, the anterior rim of the septum could be sutured to the right of the pulmonary veins thus completely correcting the totally anomalous return (Fig. 6).

In the 92 patients operated upon, with a left-to-right shunt in the atrium who form the subject of this review, including both atrial defect and anomalous venous drainage, there have been 10 in whom along with an atrial septal defect one or more pulmonary veins drained directly into the right atrium, including the one mentioned above in which all veins drained to the right. In 3 instances the superior and middle lobe veins were the only anomalous ones, in 3 the inferior vein alone and, in the other 4 cases, all right pulmonary veins entered the right atrium. When anomalous veins enter the atrium adjacent to a vena cava it is important to place an initial suture under direct vision and then be certain the cava is not obstructed, possibly by releasing the caval occlusion.

Two of these 10 patients died of conditions probably unrelated to the anomalous pulmonary venous return. In one (Case 2), there was extensive

preoperative thrombosis of the pulmonary arteries with thrombus in the atrium and severe pulmonary hypertension. The other patient (Case 4), previously reported,[2] had been treated earlier by a closed method, at which time

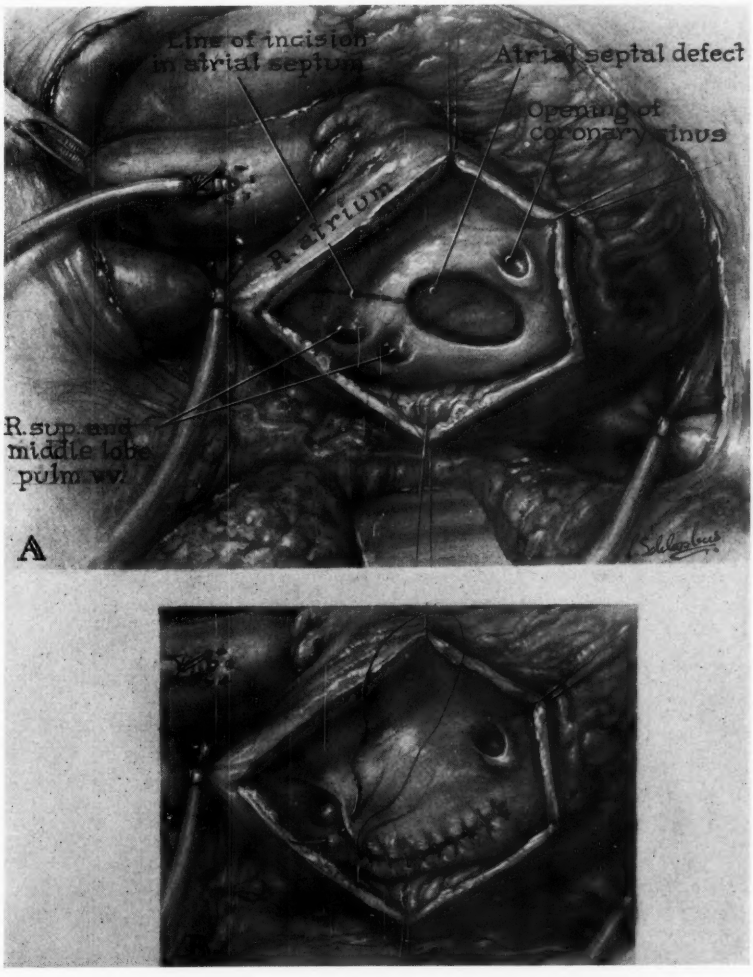


Fig. 5.—Anomalous pulmonary return to the right of an atrial septal defect. *A*, Incision of the adjacent septum is done posteriorly so that the flap of septum created can be readily sutured anterior and to the right of the entrance of the pulmonary veins (*B*).

it was not possible to correct completely the anomalous return of the inferior pulmonary vein; in addition, she developed infection about the sutures. At the second attempt to repair the defect, this time under hypothermia, fibrillation occurred which was reverted to normal rhythm but there was poor cardiac

recovery and later death. Later analysis of blood drawn for pH before the period of circulatory occlusion demonstrated a profound acidosis. We do not have an adequate explanation for this event, the only time in our experience in which such acidosis has occurred.

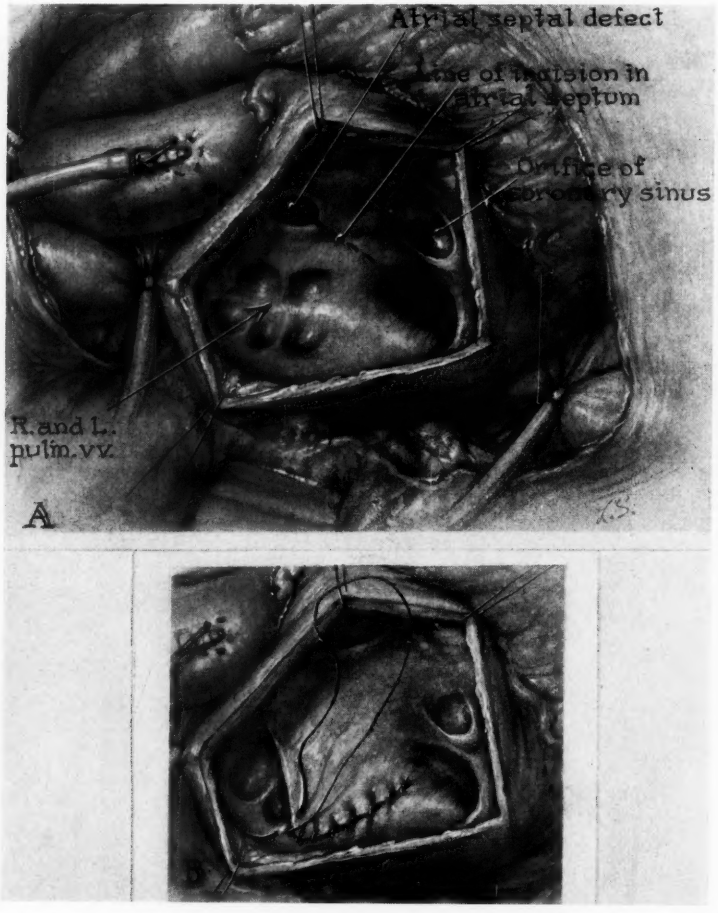


Fig. 6.—Case 1. Total anomaly of the pulmonary veins to the right of the atrial septum with a 1 cm. foramen ovale. Complete repair was effected by incision of the septum and suture to the right atrial wall (*B*).

Right Pulmonary Veins Into Right Superior Cava (Table I).—Fifteen patients have been treated with anomalous drainage into the right superior vena cava, in 5 of whom all pulmonary veins drained anomalously. The first (Case 21) was explored on the right, in 1949. After visualization of the abnormality the chest was closed with the intention of later anastomosis of the left venous trunk to the left atrium. She died in the postoperative period. The second of

these (Case 22) was not fully diagnosed until autopsy, the foramen ovale having been sutured through the atrial well. The third (Case 23) had a total anomaly with all of the veins draining into a common pulmonary venous chamber which emptied into the superior vena cava near the entrance of the

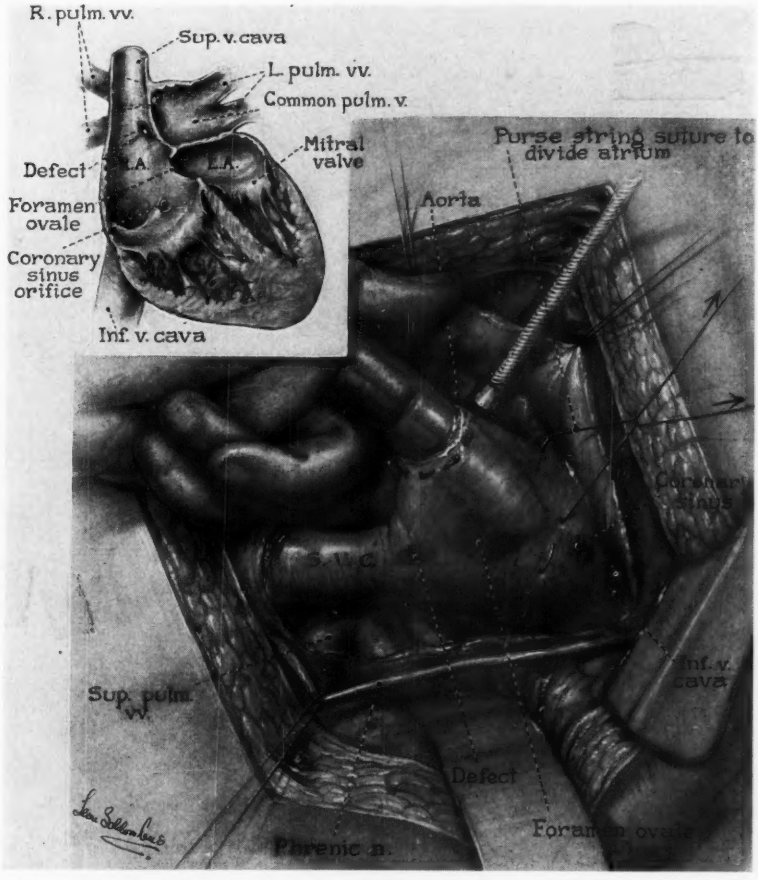


Fig. 7A.—Case 22. Drainage of all pulmonary veins to a common vein entering the superior vena cava. Attempted correction by closed procedure and a purse-string suture directing the superior vena caval and pulmonary flow through the septal defect to the left atrium.

azygos vein (Fig. 7 *A*). He was treated by a closed method in which a purse-string suture was used to approximate the caudal edge of the defect to the lateral wall of the right atrium, and the superior vena cava was partially obstructed above the point of entrance of the pulmonary veins (Figs. 7 *A* and 7 *B*). By this means, the pulmonary blood was directed to the left atrium along with a partial return from the superior vena cava. Obstruction of the superior

vena cava thus required development of collateral vessels from the superior to the inferior vena cava. Except for the patient's unwillingness to lie flat in the postoperative period and the development of superficial venous collaterals there was no evident detriment to him as a result of this procedure. Catheterization 30 months later, however, showed incomplete correction of the abnormality.

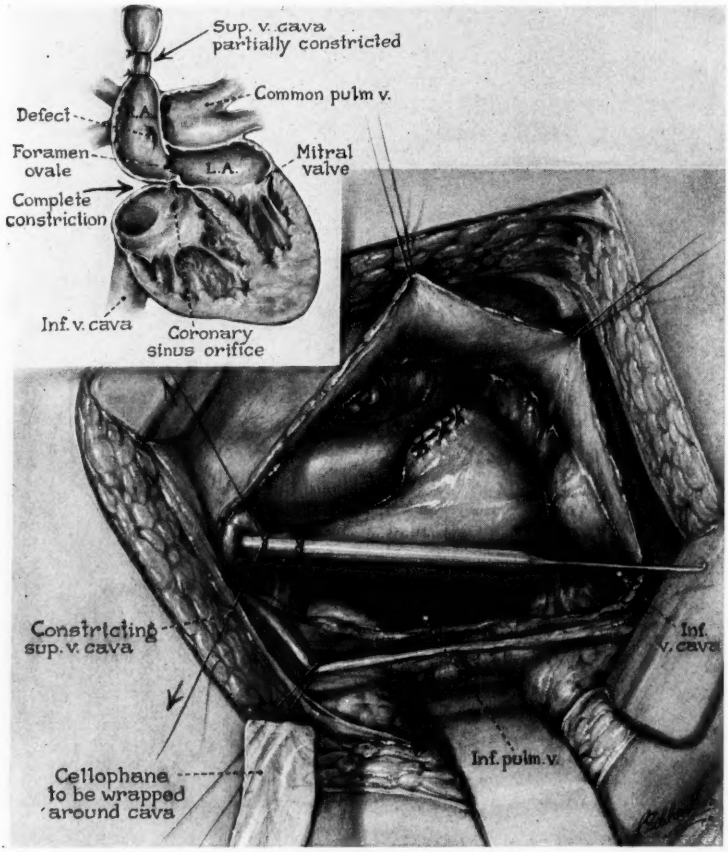


Fig. 7*B*.—Correction completed. The superior vena cava was constricted to reduce the amount of systemic venous blood entering the left atrium.

In subsequent patients, either with a partial or complete anomaly, the superior vena cava has been partitioned so that pulmonary venous blood is directed through the septal defect into the left atrium and the orifice of the superior vena cava is directed anteriorly into the right atrium. After exploratory palpation of the defect the finger has been removed and the sutures for the partition placed from the outside. When suturing in the vena cava has

been completed the venae cavae are occluded and the repair completed under direct vision (Fig. 8). In all cases, the septal defect has been a high one near the orifice of the superior vena cava.

This partitioning imposes some obstruction to the superior caval drainage but this vessel in association with anomalous return has been unusually large and adequate room was usually available. Pressures have been measured in both right and left atria and the superior vena cava after surgical partitioning and usually show an increase of 5 to 12 mm. Hg in the superior caval pressure above that in the right atrium. In only one instance has a significant complication arisen as a result of the caval sutures, the development of a chylothorax, presumably as a result of later further obstruction of the superior cava by thrombosis. All patients treated by the partitioning method survived.

Two patients with drainage of all pulmonary veins into the right superior vena cava have been treated by partitioning the superior cava and directing the pulmonary venous blood through the high atrial defect with good result.

A logical and simple solution to this defect would seem to be excision of the partition between the pulmonary venous chamber and the left atrium and closure of the resulting larger atrial and superior caval defect. Although this would have been possible in 3 of the 5 patients, it would seem impossible in the other 2 as the pulmonary artery coursed between the pulmonary venous chamber and the left atrium (Fig. 9).

In 2 patients (Cases 18 and 19), the fortunate combination of anomalous drainage into the right superior vena cava and the persistence of a left superior vena cava allowed obstruction of the right cava with impunity. In both of these instances the rim of the atrial defect was simply sutured to the anterior edge of the superior cava, thus directing all of the caval blood to the left side, and the right superior cava was ligated cephalad to the entrance of the anomalous veins but caudad to the entrance of the azygos vein (Fig. 10).

All Pulmonary Veins Draining to Coronary Sinus (Table I).—Total anomalous pulmonary venous drainage into the coronary sinus has been seen in 3 patients (Cases 26, 27, 28). In all of these, the anomaly could be easily corrected under direct vision by excision of the partition between the coronary sinus and the left atrium and closure of the single atrial defect, in essence, converting the double-barreled opening into a single one, the coronary sinus orifice being unusually large (Fig. 11). In one of the patients, a sufficient portion of the septum was not excised at the initial attempt and systemic hypotension with elevation of the pulmonary venous pressure was noted when the cardiopulmonary flow was restored. The bypass was again used and an additional portion of the septum removed so that ultimately there was little gradient of pressure between the pulmonary veins and the left atrium. Two of the patients have obtained a good result from the procedure. The third patient was an infant in whom the true state of the anomaly was not appreciated before operation and an attempt to repair the atrial defect was done under hypothermia. It was recognized that the left pulmonary veins drained into the coronary sinus but mistakenly thought that the right veins drained into the right. After 8 minutes of exploration under direct vision with caval

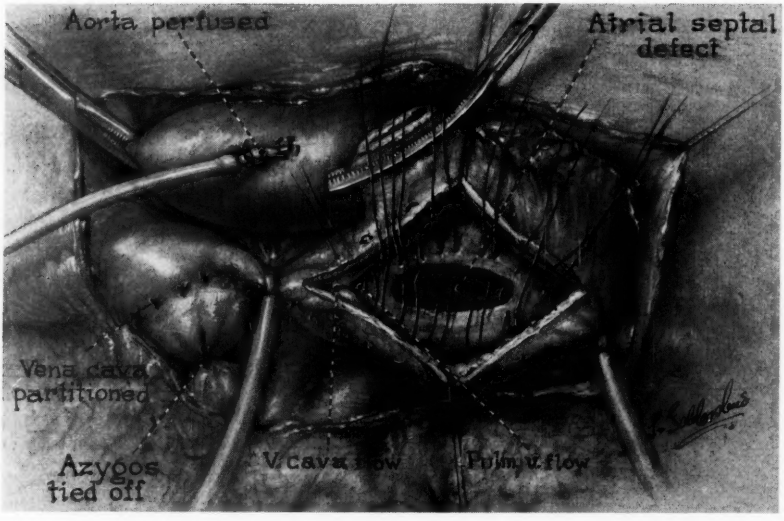


Fig. 8.—Currently employed method for correction of drainage to superior vena cava. The superior vena cava is partitioned, the pulmonary drainage being posterior and the systemic drainage anterior. After partitioning into the upper part of the atrium the repair is completed under direct vision and inflow occlusion with coronary perfusion.

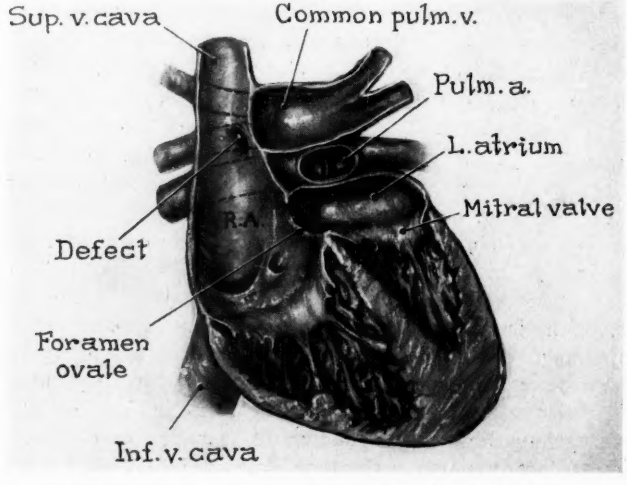


Fig. 9.—Anomaly present in 2 of the 5 cases with total drainage into the superior vena cava. The passage of the right pulmonary artery between the atrium and the common pulmonary vein would preclude excision of the septum between them in the repair.

occlusion, the circulation was re-established with the intention of a second period of occlusion for repair of the defect. Cardiac action deteriorated, however, and repair was not possible, the child subsequently dying.

In one case, an unusual atrial defect led to the preoperative suspicion of anomalous return into the coronary sinus, largely because of dilution of contrast material in the lower end of a left superior vena cava. At operation

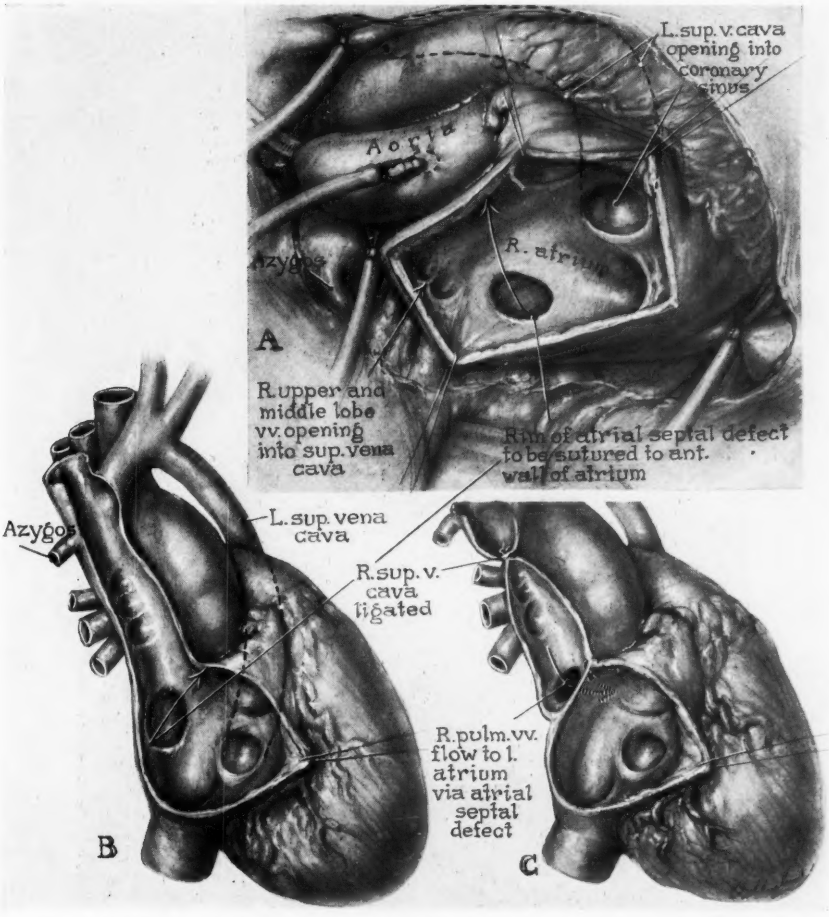
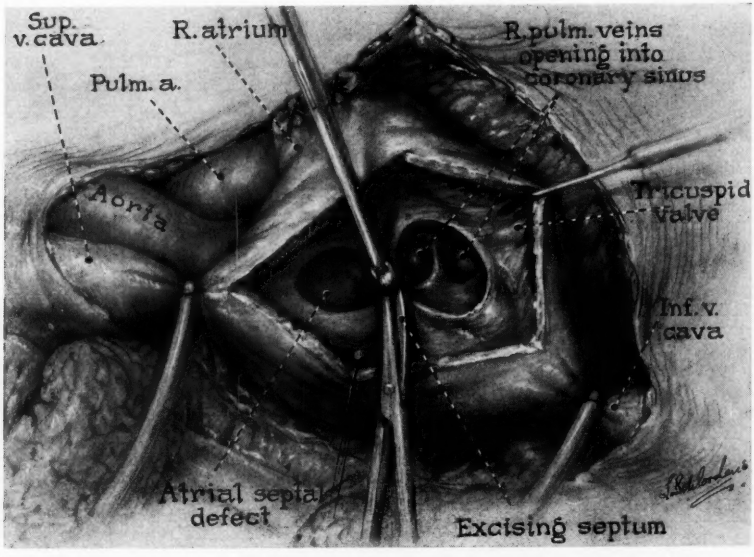

L. sup. v. cava opening into coronary sinus
Aorta
Azygos
R. atrium
A
R. upper and middle lobe vv. opening into sup. vena cava
Rim of atrial septal defect to be sutured to ant. wall of atrium
Azygos
L. sup. vena cava
R. sup. v. cava ligated
R. pulm. vv. flow to l. atrium via atrial septal defect
B
C

Fig. 10.—Cases 18 and 19. Drainage of the right lung into the superior vena cava, with persistent left superior vena cava. The right superior cava was ligated without detriment and the pulmonary venous drainage directed through the atrial defect to the left side.

during cardiopulmonary bypass, a defect was found between the left atrium and the large coronary sinus (Fig. 12). The pulmonary veins drained normally. The absence of right-to-left shunt and peripheral arterial unsaturation, cardiomegaly, and great right atrial enlargement should lead to the correct diagnosis in such a case.



A.

B.

Fig. 11.—*A*, Total pulmonary venous drainage to the coronary sinus. Tissue between the coronary sinus and the left atrium was excised.

B, The resulting larger atrial septal defect closed in the usual fashion.

All Pulmonary Veins Entering Left Superior Cava (Table I).—One of the commonest total anomalies has been that in which all pulmonary veins drain into the persistent left superior vena cava. Seven patients have been operated upon for this anomaly, the first unsuccessful attempt having been reported in 1950.[9] In 4 patients, an attempt was made to anastomose the left atrium or its

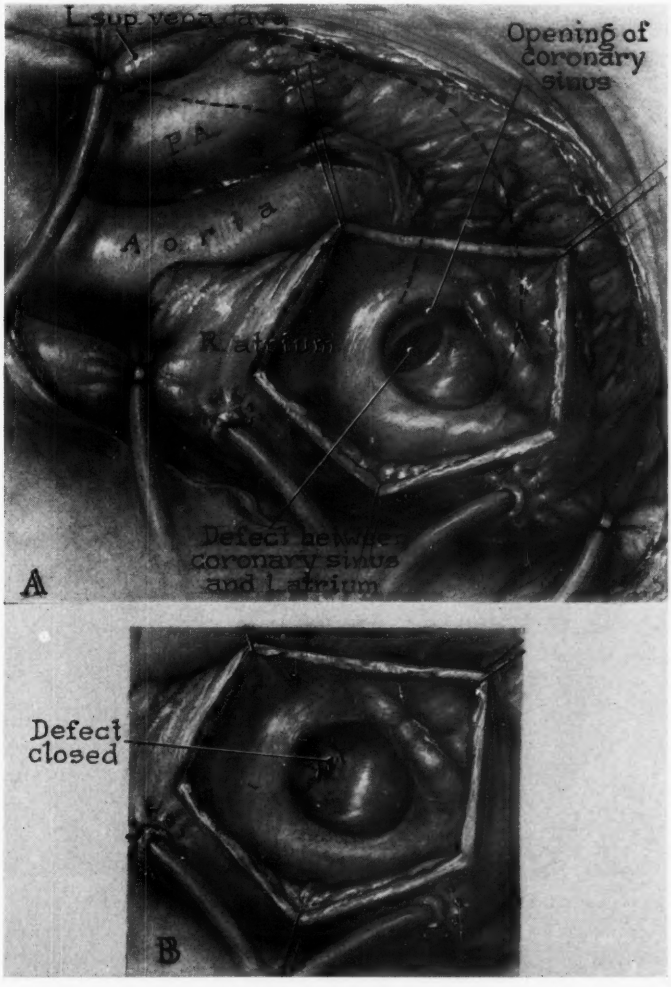


Fig. 12.—Unusual atrial septal defect between the coronary sinus and the left atrium initially diagnosed as anomaly of the pulmonary venous return. Repair effected by simple suture under direct vision with cardiopulmonary bypass.

appendage to the left superior vena cava with subsequent partial or complete obstruction of the left superior vena cava craniad to the anastomosis. In all instances, the patient died as a result either of pulmonary edema or an insufficient flow of blood to maintain systemic arterial pressure. It is believed that

the fundamental deficiency in all these operations was an insufficiently large anastomosis to the small left atrium. For this reason, after success in treatment of patients with anomalous drainage into the right superior vena cava, it seemed logical to attempt repair by diversion of the right superior vena cava through the septal defect to the left atrium with ligation of the systemic venous tributaries to the anomalous venous trunk (Fig. 13). This has been attempted in 3 patients, the last being successful.

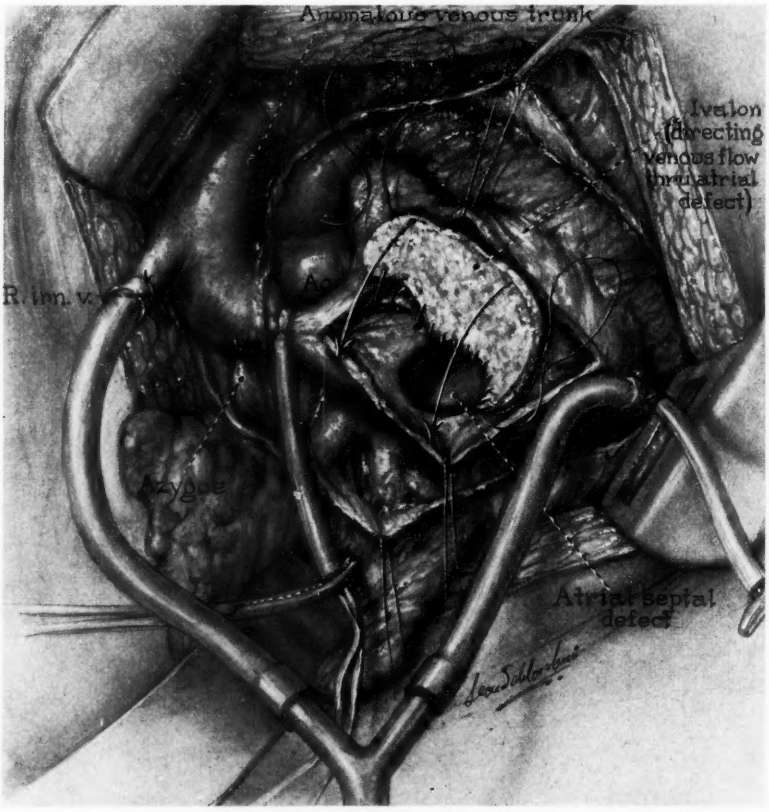


Fig. 13.—Case 35. Total pulmonary drainage to a persistent left superior vena cava. With cardiopulmonary bypass the right superior caval flow was directed by means of an Ivalon patch through the atrial septal defect to the left atrium. The right innominate and azygos veins were interrupted so that pulmonary and left innominate venous blood returned to the left atrium on completion of the repair.

Essentially the same procedure has been attempted in all 3 patients. The first, done with inflow occlusion under hypothermia, failed because of improper suturing in a small infant. The second, no larger, and in severe failure died soon after an operation considerably prolonged because of oozing. Pulmonary edema was transiently present, probably due to overtransfusion. The

third patient was an adult with cardiac enlargement and progressively limited activity. Thoracotomy through the fourth right interspace gave good exposure. The superior vena cava was cannulated through the right innominate vein, which was later divided, and the inferior cava through the nearby atrial wall. Blood from the venae cavae was led through the cardiopulmonary bypass and returned to a femoral artery. An Ivalon patch was sutured to direct the superior caval flow, with the entire pulmonary drainage, into the left atrium through the septal defect. The azygos vein was ligated, thus leaving only the left innominate vein draining with the common pulmonary trunk to the left. He has done well since discharge, has barely perceptible cyanosis, and has had striking reduction in heart size.

DISCUSSION

Of the anomalies of the pulmonary venous return, the ones least troublesome in surgical treatment are those in which there is drainage into the right auricle. These can be readily handled by methods outlined, simply transposing the veins during repair. On the other hand, such anomalies pose a considerable problem to the perfectionist in diagnosis as, with available methods, it is difficult to demonstrate the anomalous connection, sometimes even with external exploration.

Although our results in treating patients with anomalous return into the superior vena cava seem to justify continuing with this method of repair, we are not certain that as good or a better method might be the use of a prosthetic material to construct a partition in the superior vena cava.[15] This would require a greater period with the heart open and thus require the use of a cardiopulmonary bypass. Our reasons for continuing with the method described, in addition to the satisfactory results, have been twofold. First, one of the striking features in our experimental use of Ivalon was the scarring and contraction which occurred after the lapse of time. This would seem an undesirable feature in this critical area of pulmonary venous drainage, especially in a growing child. The second objection has been the greater risk, time involved, and magnitude of the operative procedure when the cardiopulmonary bypass is used as compared with inflow occlusion under hypothermia. Simplification of bypass equipment will remove this objection to its use, but we believe that partitioning of the superior vena cava without a prosthesis may still be the method of choice.

A number of attempts have been made to treat total drainage into the left superior vena cava, one of the commonest of the total anomalies. Only two have been successful.[6, 16] Both of these were done with cardiopulmonary bypass and anastomosis of the pulmonary venous trunk to the back of the left atrium through an incision in the right atrium and atrial septum. The end result of this method is preferable to the method described here if an adequate anastomosis can be obtained. Technically the method described here may be easier. Further comparison of the two procedures must be made, but either is

superior to anastomosis of the venous trunk to the left atrial appendage as the waist of the latter is often smaller than the diameter of the venous trunk.[14] Obstruction to pulmonary venous drainage must be prevented at all costs.

Use of moderate hypothermia, inflow occlusion, and coronary perfusion for the repair of atrial septal defects has been satisfactory in our hands and we have not been embarrassed by anomalies of the venous return either unsuspected or diagnosed before operation. We continue to use the technique because of its safety and simplicity, at least for the present, unless we suspect anomalous drainage other than into the right atrium or superior vena cava. For other defects and in infants we prefer the added time for exploration and treatment which cardiopulmonary bypass allows.

SUMMARY

This report concerns 35 cases of anomalous pulmonary venous drainage into the right side of the heart, in 16 of which the pulmonary drainage was totally to the right. This represents 38 per cent of the 92 patients treated during this time because of left-to-right shunt at the atrial level.

Partial anomalous drainage into the right atrium can be readily treated with modification of the techniques used in repair of the simple atrial septal defects. For this, at least for the present, we use hypothermia, inflow occlusion, and suture under direct vision and have been satisfied with the method in treating 10 patients, in one of whom all pulmonary veins entered the right atrium.

When drainage is into the right superior vena cava, the cava has been partitioned to direct the pulmonary venous blood to the left side through the atrial defect which is then closed under direct vision with inflow occlusion and hypothermia. All 11 patients treated by this method have obtained a good result.

Cardiopulmonary bypass has been used in treating successfully 2 patients with total pulmonary drainage into the coronary sinus; one treated under hypothermia died, partly because the correct diagnosis was not made before death. Bypass has been used also to treat 2 patients with total pulmonary drainage into the *left* superior vena cava, the one adult getting a good result. In these two conditions, cardiopulmonary bypass is considered essential to allow adequate time for exploration and treatment, a conclusion supported by poor results with other methods used in earlier cases.

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DISCUSSION

DR. ALTON OCHSNER, Jr., Houston, Texas.—I would like to add briefly to the fine discussion of Dr. Spencer the experience we have had in Houston since the pump-oxygenator has been available. Specifically, I would like to describe the method he referred to for total correction of that anomalous venous return which goes into the systemic system via the left superior vena cava.

(slide) This shows the total number of operations for anomalous venous return since we have had the pump-oxygenator. The group I am most interested in discussing today is the supracardiac group, that is, the pulmonary venous return comes into the systemic venous system above the heart via the left superior vena cava.

(slide) The operation that we have performed employs total cardiopulmonary bypass. Through a right atriotomy, we have extended the atrial septal defect, and then in the posterior

wall of the right and left atrium we have made a transverse incision into the common venous chamber which lies directly behind the atrium. Here you see the line of incision, here you see the opening.

(slide) Anastomosis is then made between this common venous chamber and the atrium utilizing this opening. Next, the atrial septum is swung to the right of the opening and sutured to the right posterior atrial wall. This enlarges the left atrium and swings the anastomosis into the left atrium. The atriotomy is closed and the final step is permanent ligation of the left superior vena cava.

This has been a very difficult group to repair previously, and this allows total correction of this anomaly at one operation.

(slide) We have seen only 4 cases in which the anomalous drainage was via the left superior vena cava. The first was an adult. The patient developed cardiac arrest when the chest was opened; we were unable to resuscitate him and he died. With the next 3, as you see, we have been successful. We have had 2 infants and one small child. In one, there was transient pulmonary edema, in one there was some pleural effusion. All of them are alive and well today.

DR. J. L. EHRENHAFT, Iowa City, Iowa.—As the experience with open intracardiac surgery increases, an increasing number of sometimes unsuspected associated anomalies are encountered. Our experience up to date with total and partial anomalous venous return consists of 12 patients. Of those, one patient had anomalous venous return of the right lung without an associated interatrial septal defect and one had total anomalous venous return by way of a left superior vena cava. I would like to point out that the approach to and treatment of partial and total anomalous venous return is quite different. Those two anomalies are two different things and should not be confused. Partial anomalous venous return of the right pulmonary veins entering either the superior vena cava or inferior vena cava is very common and is frequently encountered during repair of interatrial septal defects. We would like to show you a very simple method of repair for anomalous pulmonary venous connections of the right lung entering the superior vena cava.

All procedures are done under hypothermia. A modified atrioseptopexy was carried out, shunting the pulmonary venous return and, temporarily, the superior vena caval return through the interatrial septal defect. Next, the superior vena cava was transected between clamps above the entrance of the pulmonary veins into the superior vena cava. The lowermost portion of the superior vena cava was used as a channel for the pulmonary venous return while the upper segment of the transected vena cava was re-implanted into the right atrial appendage. In this manner, both venous streams were separated without obstruction of the superior vena cava or the pulmonary veins. This modification has definite merit in cases where the interatrial septal defect lies extremely high or when the superior vena cava is relatively narrow as it is often in children, preventing a splitting of the superior vena cava as has been demonstrated previously.

We would also like to show you the type of repair carried out on the patient with a total anomalous venous return. This was encountered in a 10-year-old boy. The procedure was carried out under hypothermia. The total anomalous venous return drained into a common posterior venous chamber which connected with the systemic venous return via a left superior vena cava. A large interatrial septal defect was present. The left atrium and left ventricle, even though smaller than normal, were normally developed. Complete correction of this total anomalous venous drainage was accomplished on May 1, 1957. The posterior pulmonary venous chamber was anastomosed to the left atrial appendage with a wide opening. The left superior vena cava was interrupted, diverting all pulmonary venous return through the atrial anastomosis into the left atrium. The interatrial septal defect was closed under direct vision during temporary obstruction of all inflow and outflow tracts of the heart. The patient made an uneventful postoperative recovery and is perfectly normal now.

DR. ROLLINS HANLON, St. Louis, Mo.—I would like to compliment Dr. Spencer on this presentation, which I enjoyed very much. I want to emphasize certain principles in

managing patients in the category, referred to by Dr. Ochsner, in which all the venous return from the lungs goes into the superior vena cava. As was mentioned, these are very difficult problems to manage. We have used successfully the technique described by Dr. Spencer in his first 4 patients, anastomosing the common pulmonary venous return to the left auricle. It has been said that the left auricle is too small to accept the pulmonary venous return and that this is inherently an unsatisfactory technique. We are not convinced that the technique is necessarily at fault, and we feel that with meticulous attention to details of managing the pulmonary problems, one may achieve a satisfactory result by an extracardiac operation in which the pulmonary venous return is shifted to the left auricle.

(slide) We employed this technique in an infant, aged 5 months, whose aortic pressure was 65 mm. Hg and peripheral arterial oxygen saturation was 91 per cent. As you can see from the diagram, all venous return drained into the left anterior cardinal vein area. At operation we unwisely closed the atrial septal defect first. The heart immediately behaved poorly and it was necessary to carry out anastomosis by an extracardiac technique of the pulmonary veins to the left auricle. We did not use cardiopulmonary bypass originally during this anastomosis but, because we had to dislocate the heart so strikingly, it was essential to pull off blood from the venae cavae, which were blocked, by turning the heart forward. This permitted a one-centimeter anastomosis to the auricle and this infant is now doing well at 19 months.

The point I would like to emphasize is that the loss of many of these patients with so-called pulmonary congestion may be preventable by use of tracheostomy and intensive efforts at clearing secretions from the tracheobronchial tree. Assisted respiration with a mechanical ventilator is also useful at times; this infant had mechanical support of ventilation through a tracheostomy for a week before we considered him out of danger. At times during this period, the entire lung field showed severe congestion and patchy atelectasis, except for one upper lobe. Without the most intensive care, such patients will die of respiratory inadequacy to which some residual difficulty with pulmonary venous drainage may contribute significantly. Establishment of the connection between pulmonary vein and left auricle by a transatrial approach may be more satisfactory than the extracardiac approach used in our patient, since it may permit a larger anastomosis and also enlargement of the left auricle by internal reconstruction.

DR. ROBERT P. GLOVER, Philadelphia, Pa.—The ingenuity shown by Dr. Spencer and Dr. Bahnson in the handling of the complicated cases in their series is admirable. We have felt that most of the cases encountered in which an atrial septal defect is complicated by anomalous venous drainage can be handled correctly by simple "closed surgical" techniques. For the most part, when partial anomalous venous drainage is present, the atrial septal defect is of the "secundum" type. Under these circumstances, the septal defect can easily and permanently be closed by the circumferential suture introduced by Sondergaard and further developed by Björk. Re-implantation of the venous anomalies, if this proves to be necessary, can also be carried out quite as easily by closed anastomotic methods as by open.

Four recent cases exemplify this contention.

(slide) In two instances, the venous drainage from the upper and middle lobes on the right entered the right atrium in a common trunk well below the orifice of the superior vena cava at a point just opposite the interatrial septal defect. By carrying the encircling suture anterior to this faulty site of entrance into the right atrial wall itself (not as normally in the interatrial groove), the septal defect was tightly closed and the venous return re-directed into the left atrium without resort to anastomotic measures. One of these patients, as subsequent events proved, should not have been operated upon by any technique, for she died 3 weeks later due to an irreversible pulmonary vascular resistance of high degree.

(slide) In the third case, three veins from the right upper and middle lobes entered the superior vena cava just above the right atrium. An ellipse of cava wall was excised containing all three veins and was re-implanted into the left atrium with one anastomosis. The septal defect was easily closed by tightly tying the previously placed circumferential suture without compromising the lumen of the transplanted vessels. The result was excellent.

(slide) A fourth case was somewhat similar to the third except that the three veins entering the superior vena cava were well above the right atrium and could not be stretched to the left atrium. A freeze-dried segment of an aortic arch was used to bridge the gap. The largest of the three vessels was anastomosed to the arch itself end-to-end, the next largest vessel to the carotid stump end-to-end, and the third (very small) was simply ligated. The distal end of the graft was then attached to the left atrium, anastomosing the full circumference of the aorta to the incised left atrium. The result was likewise most satisfactory.

Familiarity with the Sondergaard technique and its many possible modifications makes an approach such as described altogether feasible, safe, and satisfactory, without resort to more complicated methods.

DR. SPENCER (Closing).—I have nothing further to say, and I would like to thank the discussers for their interesting comments.

PULMONARY RESECTION IN THE TREATMENT OF TUBERCULOSIS

Experience With 1,730 Patients

Raymond J. Barrett, M.D. (by invitation), Hunter S. Neal, M.D. (by invitation), J. C. Day, M.D., Paul T. Chapman, M.D. (by invitation), Paul V. O'Rourke, M.D., E. J. O'Brien, M.D., and William M. Tuttle, M.D., Detroit, Mich.

THE ADVENT of chemotherapy has brought about profound changes in the treatment of pulmonary tuberculosis during the past decade. The expansion of the area of usefulness of surgical therapy, made possible by these changes, has been so rapid that definition of its limits has become increasingly difficult. The surgeon and his medical colleagues alike have been perplexed by conflicting evidence purporting on the one hand to support its further expansion and on the other to indicate the need for contraction of its scope.

The extensively involved lungs of the very ill often have miraculously survived the initial infection with clearing of reversible components of disease, leaving behind permanently damaged tissue, the removal of which offers the only hope for survival and rehabilitation. At the other end of the spectrum, the antimicrobial agents have been found so successful in the control of lesser amounts of disease that the area where surgery is indicated is not sharply defined.

The accumulated experiences of many individuals and the cooperative studies of the VA-Armed Forces have done much to define the role of resection but many questions remain unanswered. Only by critical appraisal of the results of a significant number of these procedures can any conclusions be reached and some of the questions be answered. That any surgical procedure must be judged by its results, and measured against the hazards involved in its employment, is a truism. When resection is employed in pulmonary tuberculosis, one must be certain that the morbidity and mortality attendant upon it is not excessive and that it offers a good chance for rehabilitation of the patient. Equally important is the obligation to the community to which the patient is returning. The patient must not be a menace to the community or his family because of infectiousness of sputum.

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Read at the Thirty-eighth Annual Meeting of The American Association for Thoracic Surgery at Boston, Mass., May 16-18, 1958.

It is the purpose of this report to present an analysis of our experience with pulmonary resection with particular reference to mortality, morbidity, the incidence of relapse, and the ultimate fate of the patient. With the correlation of various factors such as extent of disease, type of operation, infectiousness of sputum, age, race, and sex, complications, mortality, and the present status of the patients, it is hoped that some helpful information may be obtained.

Perhaps certain disclosures may be forthcoming which will offer some help in defining more clearly the areas where resection can be employed safely and effectively and, more important, the areas where resection should be avoided because of excessively high risk.

HISTORICAL SURVEY

Although sporadic efforts were made to excise portions of lung before the turn of the century following the tragic experience of Block, resections were successfully accomplished by Tuffier in 1891 and Lowson in 1893. However, the first planned lobectomy for tuberculosis was done by Freedlander[1] in 1934. The following remarks concerning early experiences with resection in pulmonary tuberculosis are quoted from Jones'[2] historical review in the John Alexander Monograph Series[2]:

"In 1935, Eloesser and O'Brien each reported a case of one-stage lobectomy for tuberculous cavities; both patients died of a tuberculous spread to the remaining lobes of lung. In 1934, Beye inadvertently but successfully resected the right lower lobe of the lung for a saccular bronchiectasis, which on microscopical study proved to be a tuberculous cavitated lung which had failed to heal following multiple thoracoplasties. The latter patient had a bronchocutaneous fistula when last reported. In 1936, Lindskog had by accident performed a left pneumonectomy on a tuberculous bronchiectatic lung, and a year later the patient was still febrile with fistula, a tuberculous infiltration and active disease with localized empyema. In 1938 Jones and Dolley reported their original series of five resections for tuberculosis, one of a number of small series of tuberculosis resections reported by several surgeons. And finally in 1940, at the meeting of the American Association for Thoracic Surgery in Cleveland, there was a lengthy discussion on the subject, several contributors reporting their own limited series and results. It was obvious at this time that because of the complications of tuberculous infiltration spreads, bronchial fistula, empyema and toxic pleuritis, most surgeons had lost their enthusiasm for resection in tuberculosis. At this meeting the collective series of the several contributors might be summarized in the following fashion: among the resections, there were nineteen cases of tuberculosis treated by total pneumonectomy, only three patients were considered well, six improved, and two were failures. Eight of the nineteen patients were dead, a pneumonectomy mortality of 40.2 percent. At the same time, the various participants in the discussion had a collective series of thirty-one lobectomies for tuberculosis, sixteen patients were classified as well, six as improved, and one as uncertain. There were eight deaths reported, a lobectomy mortality of 20.5 percent. It was significant that in ten of the sixteen patients considered well, tubercle bacilli were not found in the sputum before the lobectomy was performed. The implication, however, need not necessarily

be made that these were cases who did not have active tuberculosis, for it is doubtful if any of these patients ever had gastric or sputum cultures and animal inoculations before their surgery."

There followed a period of pessimism and discouragement because of the high incidence of complications and formidable mortality rate. A new impetus was given to resection by the work of Blades and Kent,[3] and the views expressed by Churchill[4] concerning the feasibility of the individual ligation technique, later popularized by Overholt,[5] are now universally accepted and the technique employed.

The last barrier to the successful employment of resection in pulmonary tuberculosis was removed with the discovery of the antituberculosis drugs. During the past decade, resection has been accomplished with increasing frequency and safety and has largely replaced other forms of surgical treatment.

CLINICAL MATERIAL

The statistical data in this report are based upon our experience with pulmonary resection in tuberculosis during the period from Nov. 1, 1949, to Dec. 31, 1957. Since the initial report on the first 202 resections in our series, an additional 1,567 operations have been performed upon 1,528 patients. These original operations are not included in this report except that they appear in the total number of operations.

A brief résumé of the results of the first 202 resections reveals an operative mortality of 6.9 per cent and a late mortality of 5.5 per cent as of the date reported. There were 104 pneumonectomies, of which 19 of the patients are dead (18 per cent), and 98 lobectomies with 6 deaths (6.1 per cent). Bronchopleural fistula occurred in 27, or 13 per cent, of the patients, and was the major cause of death. Spread of disease occurred in 14 per cent of lobectomies and 10.5 per cent of the pneumonectomies. One hundred and seventy-seven patients were living 6 months to 5 years later and 150 (85 per cent) had negative cultures. A follow-up study of this initial group will be the subject of a subsequent report.

TABLE I. COMPOSITION OF ENTIRE SERIES BY AGE, SEX, AND RACE

AGE (YR.)	WHITE MALE (PER CENT)	WHITE FEMALE (PER CENT)	NEGRO MALE (PER CENT)	NEGRO FEMALE (PER CENT)
0-15	0.5	2.5	2.0	4.5
16-29	24.3	46.9	28.6	50.5
30-39	23.7	29.5	24.7	30.8
40-49	26.7	13.9	28.3	10.3
50-59	20.2	6.5	15.5	3.9
60-69	4.1	0.7	0.8	0.0
Total	558	447	254	311

In the group currently being analyzed, there are 1,528 patients on whom 1,567 operations were performed. There were 100 pneumonectomies, 787 lobectomies, 579 segmental resections, and 101 wedge or local excisions. There were 35 bilateral resections and 10 patients had second resections on the same side. In Table I, showing the distribution of patients according to age, sex,

and race, it will be noted that the great majority of females, both white and Negro, were under 40 years of age, there being actually 55 per cent of them under 30 years of age. The males are more evenly distributed in the middle and older age groups, 50 per cent of white males being over 40 years of age and almost 25 per cent 50 or over.

Table II, which shows the distribution according to extent of disease, reveals a very striking difference between the white and Negro groups. A very high percentage of both sexes in the Negro group had far-advanced disease. Thus, it is seen that there are four rather distinct groups. The young white females have predominantly moderately advanced disease. The young Negro females have predominantly far-advanced disease. The white males are middle-aged and have predominantly moderately advanced disease, and the Negro males are middle-aged and have far-advanced disease.

TABLE II. TYPE OF DISEASE BY RACE AND SEX

	WHITE MALE (PER CENT)	WHITE FEMALE (PER CENT)	NEGRO MALE (PER CENT)	NEGRO FEMALE (PER CENT)
Primary	0.2	0.2	1.6	1.0
Minimal	7.8	8.1	2.4	4.8
Moderatey advanced	53.0	55.3	37.4	33.0
Far-advanced	39.0	36.4	58.6	61.2

When the present status is related to sex and race, as is shown in Table III, it is seen that the females do far better than the males in both the white and Negro groups, 87 to 89 per cent of them being in the inactive group as contrasted to the white males, only 76.3 per cent of which are inactive, and the Negro males, 82.2 per cent of which are inactive. There are six times as many white males as white females in the active status. More white males are dead than white females. The over-all mortality rate is lowest in white females (2.4 per cent) and highest in Negro males (8.0 per cent). Sixty, or 3.8 per cent, are classified as active, and 1,293, or 82.5 per cent, as inactive. It will be noted that these percentages relate to the total in the series and not just to the survivors.

TABLE III. PRESENT STATUS BY RACE AND SEX

	WHITE		NEGRO		
	MALE	FEMALE	MALE	FEMALE	TOTAL
Active	40 (7.2)	6 (1.2)	8 (3.1)	6 (1.9)	60 (3.8)
Inactive	425 (76.3)	386 (86.7)	206 (82.2)	276 (88.8)	1,293 (82.7)
Dead	33 (5.9)	11 (2.4)	20 (8.0)	11 (3.6)	75 (4.8)
Unknown	60 (10.6)	44 (9.8)	17 (6.8)	18 (5.8)	139 (8.6)

The total mortality figures and present status of activity are found in Table IV, listed according to type of operation. The operative mortality was 2.9 per cent, and the nonoperative mortality was 1.9 per cent, or a total of 4.8 per cent. The former represents any deaths related to operation, however far removed from it, and coincides with hospital deaths. The late deaths were those occurring subsequent to discharge from the hospital, including all deaths from any cause.

TABLE IV. TYPE OF OPERATION VERSUS PRESENT STATUS

	ACTIVE	INACTIVE	DEAD		UNKNOWN	TOTAL
			OPERATIVE	NONOPERATIVE		
Pneumonectomy	4	67	12	4	13	100
Lobectomy	38	633	24	15	77	787
Segmental	15	507	10	8	39	579
Wedge	3	86	0	2	10	101
Total	60	1,293	46	29	139	1,567
Percentages	3.8	82.5	2.9	1.9	8.9	100

An interesting paradox is seen in the pneumonectomy group (Table V), as it relates to the large percentage of Negro females. Whereas they make up only one fifth of the total series, they comprise a third of the pneumonectomy group. They are also conspicuous for the absence of mortality, either operative or late, as contrasted to an extremely high mortality rate in the Negro males (30.7 per cent), and a high mortality rate in both sexes in the white group. This very striking phenomenon is unexplained by any other factor than youth since only 36 per cent of Negro females who had pneumonectomies were beyond the age of 30, as contrasted to 66 per cent of white females, 77 per cent of Negro males, and 93 per cent of white males. Actually, 43 per cent of all white males who had pneumonectomies were beyond the age of 50.

TABLE V. PNEUMONECTOMIES VERSUS RACE AND SEX

	WHITE		NEGRO		TOTAL
	MALE	FEMALE	MALE	FEMALE	
Active	3	0	0	1	4
Inactive	16	16	6	29	67
Dead					
Operative	4 (14.2%)	4 (15.4%)	4 (30.7%)	0	12
Nonoperative	2	1	1	0	4
Unknown	3	5	2	3	13
Total	28	26	13	33	100

TABLE VI. LOBECTOMIES BY RACE AND SEX

	WHITE		NEGRO		TOTAL	
	MALE	FEMALE	MALE	FEMALE		
Active	22	5	6	5	38	4.8%
Inactive	176	162	126	169	633	80.5%
Dead						
Operative	12 (4.9%)	2 (1.0%)	8 (5.2%)	2 (1.0%)	24	3.1%
Nonoperative	6 (2.4%)	1 (0.5%)	4 (2.6%)	4 (2.0%)	15	1.8%
Unknown	30	26	10	11	77	9.8%
Total	246	196	154	191	787	

When the lobectomies are analyzed by race and sex, a striking difference is apparent between the sexes. The operative mortality in the males is five times that in the females. This again appears to be a function of age. The percentage of lobectomies classified as inactive is 80.5 per cent.

TABLE VII. SEGMENTAL RESECTIONS BY RACE AND SEX

	WHITE		NEGRO		TOTAL	
	MALE	FEMALE	MALE	FEMALE		
Active	12	1	2	0	2.6%	15
Inactive	204	171	62	70	87.5%	507
Dead						
Operative	4 (1.6%)	3 (1.6%)	1 (1.4%)	2 (2.6%)	1.7%	10
Nonoperative	5 (2.0%)	0	2 (2.8%)	1 (1.3%)	1.4%	8
Unknown	19	11	5	4	6.8%	39
Total	244	186	72	77		579

TABLE VIII. WEDGE AND SUBSEGMENTAL RESECTION BY RACE AND SEX

	WHITE		NEGRO		TOTAL	
	MALE	FEMALE	MALE	FEMALE		
Active	3	0	0	0	3	3%
Inactive	29	37	12	8	86	86%
Dead						
Operative	0	0	0	0	0	0%
Nonoperative	0	0	0	2	2	2%
Unknown	8	2	0	0	10	10%
Total	40	39	12	10	101	

Both the segmental resections and the wedge and subsegmental group did well as regards operative and late mortality and present status. The operative mortality was 1.7 per cent in segmentectomies and there were no operative deaths in the wedge and subsegmental resections. The percentages appearing in the inactive status in these two groups were 87.5 per cent and 86 per cent, respectively, as would be anticipated.

COMPLICATIONS

The major complications of surgery were analyzed according to infectiousness of sputum in each type of operation. The total incidence of fistulas was 6.2 per cent. In patients with positive sputum, prior to operation, the rate was 11.4 per cent and, in patients with negative sputum, it was 5.1 per cent. This last figure is apparently the minimum incidence attainable under the best conditions in our series, i.e., the negative-sputum status. The incidence of bronchopleural fistula in lobectomy was 5.3 per cent but was three times as high in positive-sputum cases as in negative cases. The highest incidence of fistula was in segmental resections with positive sputum, being 17.8 per cent and somewhat higher than was anticipated, although we were prepared for a higher incidence. Sixty per cent of fistulas in the segmental resections, however, occurred in moderately advanced cases rather than in a preponderance of far-advanced, as we had anticipated in explanation of the figure. Thirty-three of the 50 fistulas in this group, however, were in white males. Thus, two thirds of the bronchopleural fistulas occurred in white males who comprised only 42 per cent of the segmental resections.

The incidence of nontuberculous empyema (bacterial empyema without fistula, predominantly staphylococcic) was 0.6 per cent and spread of disease was equally insignificant as a complication, being 0.7 per cent.

TABLE IX. COMPLICATIONS OF SURGERY VERSUS SPUTUM

	SPREAD			EMPYEMA			BRONCHOPLEURAL FISTULA		
Pneumonectomy (100)	63 + 1/63 37 – 2/37	1.6% 5.4%	3%	0 0			4/63 2/37	6.3% 5.4%	6%
Lobectomy (787)	155 + 6/155 632 – 2/632	3.9% 0.3%	1.1%	0 5/632		0.8%	18/155 24/632	11.6% 3.8%	5.3%
Segmental resections (579)	56 + 1/56 523 – 0			1/ 56 3/523	1.8% 0.6%	0.7%	10/56 40/523	17.8% 7.6%	8.6%
Wedge resections (101)	7 + 0 94 – 0			0 0			0 1/94	1.1%	1%
	+ 8/ 281 – 4/1,286	2.5% 0.3%		1/ 281 8/1,286	0.4% 0.6%		32/ 281 66/1,286	11.4% 5.1%	
Total	12/1,567	0.7%		9/1,567	0.6%		98/1,567	6.2%	

\+ Indicates patients positive prior to surgery.
– Indicates patients negative prior to surgery.

An analysis of the fate of the patients with bronchopleural fistulas, the total incidence of which was 6.2 per cent, reveals that this complication is least serious in segmental resections, although still resulting in a formidable 12 per cent mortality rate, as noted in Table X. The seriousness of bronchopleural fistula in pneumonectomy is well known. Of the six occurring in this series, 3 patients (50 per cent) are dead, and 3 (50 per cent) are active. It is also a serious complication in lobectomy, where it resulted in death in 26 per cent of the 42 patients with fistula. Its seriousness is also reflected in the 10 per cent, 19 per cent, and 50 per cent active status in segmental resections, lobectomies, and pneumonectomies, respectively. The over-all percentages for the group were 17 per cent active, 55 per cent inactive, and 20 per cent dead.

TABLE X. FATE OF BRONCHOPLEURAL FISTULA

98/1,567 = 6.2%		ACTIVE	INACTIVE	DEAD	UNKNOWN
Pneumonectomies (100)	6 BPF	3 (50%)		3 (50%)	
Lobectomies (787)	42 BPF	8 (19%)	19 (45%)	11 (26%)	4 (9.5%)
Segmental resections (579)	50 BPF	5 (10%)	36 (72%)	6 (12%)	3 (6%)
Wedge resections (101)	1 BPF	1 (100%)			
		17/99	55/99	20/99	7/99
Total	99	17%	55%	20%	7%

RELAPSE

Five of 100 patients with pneumonectomies relapsed, 3 of whom were positive prior to operation and 2 negative. In the positive group, one third are dead and one third are active, while in the negative group, one half are active.

There was a total of 51 relapses in 787 lobectomies, a rate of 6.5 per cent. The incidence of relapse in the positive cases was 3½ times that in the negative group.

The seriousness of relapse in positive patients is emphasized by the fact that in relapses occurring in positive-sputum patients, there was an 8.7 per

TABLE XI. RELAPSE VERSUS SPUTUM AND OPERATION

WHITE MALE		WHITE FEMALE		NEGRO MALE		NEGRO FEMALE	
100 Pneumonectomies							
+	−	+	−	+	−	+	−
18	10	16	10	9	4	20	13
Relapse							
2	0	0	0	1	0	0	2
11%				11%			15.4%
		+		−			
		3/63 = 4.8%		2/37 = 5.4%			
Total		1 active		1 active			
		1 inactive		1 inactive			
5/100 = 5%		1 dead					
787 Lobectomies							
+	−	+	−	+	−	+−	−
60	185	42	153	21	133	32	159
Relapse							
14	11	2	7	4	7	3	3
23.3%	5.9%	4.8%	4.6%	19%	5.2%	9.4%	1.9%
		+		−			
		23/155 = 14.9%		28/632 = 4.4%			
Total		16 active		15 active			
		5 inactive		11 inactive			
51/787 = 6.5%		2 dead		2 unknown			

cent mortality and 69 per cent of the patients are still active, as contrasted with no deaths in the negative patients with relapses and 53 per cent still active in the latter group.

In 579 segmental resections, there were 25 relapses with an incidence twelve times as high in the positive as among the negative cases. There is one

TABLE XII. RELAPSE VERSUS SPUTUM VERSUS OPERATION

WHITE MALE		WHITE FEMALE		NEGRO MALE		NEGRO FEMALE	
579 Segmental Resections							
+	−	+	−	+	−	+	−
30	214	10	176	7	65	9	68
Relapses							
8	6	2	4	4	1	0	0
27%	2.8%	20%	2.3%	57%	1.5%	0	0
		+		−			
		14/56 = 25%		11/523 = 2.1%			
Total		9 active		7 active			
25/579 = 4.3%		3 inactive		2 inactive			
		2 unknown		1 dead			
				1 unknown			
101 Wedge Resections							
+	−	+	−	+	−	+	−
1	39	5	33	0	12	1	9
Relapses							
0	2	0	0	0	0	0	0
0	5.1%	0	0	0	0	0	0
Total: 2/101 = 2%; both active							

Summary

Pneumonectomy	+ Sputum 14.6%
Lobectomy	
Segmental resections	Total: 5%
Wedge resections	− Sputum 3.3%

death (4 per cent mortality in the entire group) and the percentage still active is the same in both groups.

There were two relapses in 101 wedge resections, both of which are still active. For all types of operation, the relapse incidence was 5 per cent; however, it was over four times as great in the positive-sputum cases as in the negative, or 14.6 per cent and 3.3 per cent, respectively.

Of the total of 83 relapses, 23 patients are inactive (27.77 per cent), 51 or 63 per cent are active, and 4, or 5 per cent, are dead.

CAUSES OF SURGICAL MORTALITY

12 Pneumonectomy Deaths
- 5 Pulmonary insufficiency
- 3 Bronchopleural fistula
- 1 Massive infarct
- 1 Pulmonary edema
- 1 Massive atelectasis of opposite lung
- 1 Immediate postoperative; unexplained

24 Lobectomies
- 7 Bronchopleural fistula
- 3 Transfusion reaction and/or lower nephron syndrome
- 2 Hemorrhage
- 2 Pneumonia
- 1 Empyema
- 1 Pulmonary edema
- 1 Massive embolism to opposite main pulmonary artery
- 1 Cerebrovascular accident
- 1 Wound infection with massive hemorrhage
- 1 Cardiac arrest
- 1 Respiratory insufficiency
- 1 GI hemorrhage; 6 weeks postoperatively
- 2 Unexplained

10 Segmental Resections
- 6 Bronchopleural fistula
- 1 Pulmonary insufficiency
- 1 Spread
- 1 Hemorrhage
- 1 Myocardial infarction—clinical impression

The causes of surgical mortality, as listed above, are, for the most part, self-explanatory. It is noted, however, that the incidence of bronchopleural fistula as a cause of death, although still high, is not nearly so common a cause of death as in the initial series reported. Pulmonary insufficiency, which appears as the most frequent cause of death in the pneumonectomy group, would seem to indicate that a considerably larger group of patients is operated upon with marginal functional respiratory reserve.

REVIEW OF RESULTS OF OTHER SERIES

There is a striking similarity in the mortality, morbidity, and relapse rates and the present status of patients reported in large individual and collected series.

The experience reported by the VA-Armed Forces study group is by far the largest, since it represents the combined results of about 40 participating Veterans Administration hospitals. It is summarized as follows by Raleigh and Steele[6]: There were 5,054 pulmonary resections performed for tuberculosis between 1952 and 1956. The surgical mortality was 15 per cent in 259 pneumonectomies, 3 per cent in 1,800 lobectomies, and 1 per cent in 2,254 segmental resections. Using the incidence of postoperative empyema as an index of surgical morbidity, the morbidity was 17 per cent for pneumonectomy, 4.5 per cent for lobectomy, 4 per cent for segmental resection, and 1 per cent for subsegmental resection. They report that in a small number (18) of pneumonectomies in negative-sputum patients there was no mortality or morbidity. A 10 per cent mortality rate and 20 per cent incidence of empyema was encountered in those patients positive prior to operation. They also note that the mortality rate was almost tripled and the morbidity rate was four times as great in lobectomies in patients with positive sputum as contrasted to those with negative sputum prior to operation.

Gale, Dickie, Rankin, and Curreri[7] report a series of 1,165 consecutively operated cases from 1944 to May, 1954. One hundred and fifty-eight patients were 50 years of age or older at the time of surgery. Thirty-eight were more than 60 years old. There were 36 operative deaths (3.1 per cent); 21 following pneumonectomy, 13 following lobectomy, and 2 following segmental resection. There were 39 (3.3 per cent) nonoperative deaths. Ninety-one per cent, 1,015 of 1,115, were clinically well and inactive. Two per cent are classified as having active tuberculosis.

Robinson, Jones, Meyer, and Reding[8] reported a 12-year experience with 465 resections in which the operative mortality was 2.8 per cent and the late mortality was 2.4 per cent, a total mortality rate of 5.2 per cent. In the long-term, streptomycyin-treated patients, the mortality and complication rates were reduced to 1.7 per cent and 7.6 per cent, respectively, from 16 per cent and 26 per cent, respectively, in the pre-streptomycin period. The follow-up study included only 190 cases (144 lobectomies and 45 pneumonectomies) from the Barlow Sanatorium in which the over-all operative mortality was only 2.1 per cent of 150 patients having resection between 1943 and 1953, inclusive, and followed for 1 to 11 years. One hundred and thirty-one (94 per cent) had negative sputum. Of 104 thoracoplasty patients followed the same length of time, 85 (82 per cent) had negative sputum.

Bickford, Edwards and co-workers[9] reviewed 1,575 operations done from 1947 to April, 1956. There were 27 operative deaths for the full series, a rate of only 1.71 per cent. A 5-year follow-up on the first 399 showed a 2.25 per cent mortality and a 5 per cent fistula rate. Three hundred and forty-seven, or 87 per cent, were clinically well and 29, or 7 per cent, were active or disabled. However, there were only 19 patients over the age of 50 of which 2 died (one had had a segmental resection and one a pneumonectomy) and 4 developed empyema. Reactivation occurred in 14 per cent. The causes of death included 6 due to pulmonary embolism which, in our experience, has been

rare. They state that they have taken a more conservative approach to the caseous nodules and excise only those over 2.5 cm. in diameter. They use space-reducing thoracoplasty in everything except minor segmental resections.

Hirdes and Stegerhoek[10] report on 700 resections done between October, 1949, and February, 1954. By February, 1955, 23 of the patients had died, a mortality of 3.3 per cent. A total of nine tuberculous, and four nonspecific, empyemas were observed. The number of relapses in a follow-up of 2 to 5 years amounted to 7 per cent. On Feb. 1, 1955, 92 per cent of the patients were well.

Sir Clement Price-Thomas[11] reports that in 366 cases, there was a total of 28 deaths, a mortality rate of 7.65 per cent. There were 2 early deaths in 107 lobectomies (1.86 per cent), 4 early deaths in 194 segmental resections (2.06 per cent), and 12 early deaths in 65 pneumonectomies (18.46 per cent). In 85 patients who had open cavitation at the time of operation, 17 died in the hospital, a mortality rate of 20 per cent.

Chamberlain and Klopstock[12] state that in their own original series of 300 segmental resections, reported in 1953 and followed from 1 to 5 years, 93.7 per cent were living and well; 3 per cent were living with disease, and 3 per cent were dead. Nine out of 300 died, 3 (1 per cent) as a result of operation, and 6 (2 per cent) from postoperative complications, or as a result of secondary operations for control of complications. The major complication was bronchopleural fistula in 5.3 per cent. There were 18 exacerbations in the entire series (6 per cent). In an additional 700 patients, they indicate mortality rates and morbidity rates comparable to those in the VA-Armed Forces series which are 1 per cent and from 2 to 6 per cent, respectively.

Robinson and Corpe[13] report a series of 119 pneumonectomies which comprised 16 per cent of 746 pulmonary resections done at Battey State Hospital from 1948 to 1957. Almost all patients were operated upon in the face-down position and all were connected to water-seal drainage for 28 hours, regardless of the absence or presence of thoracoplasty. A concomitant thoracoplasty was done if the patient was in good condition. Ninety-seven were performed for "destroyed lung" and 14 for thoracoplasty failure. There were 8 surgical deaths in patients with positive sputum (9.4 per cent) and 2 deaths in patients with negative sputum (6 per cent), the total mortality rate being 8.4 per cent. The tuberculous complications in the group with infectious sputum are listed as 7 bronchopleural fistulas, 8 tuberculous empyemas, 2 of chest wall tuberculosis, 4 spreads, and 19 in which there was disease of the bronchial stump. There were 5 late deaths for an over-all mortality of 12.6 per cent.

In a recent communication, Corpe and Blalock[14] have studied a group of patients with persistent cavitation and noninfectious sputum after discharge from the hospital. The relapse rate in 159 patients has been 8.8 per cent. They had been negative for a mean average of nearly 16 months prior to discharge and have been followed for a period of 3 to 48 months (only 26 followed for less than 1 year).

The death rate was 3.8 per cent but they had not had bacteriologic or roentgenographic relapse. In a small group of patients who had refused surgery, 8.3 per cent have had reactivations. Among 38 patients in whom drug therapy was discontinued, there have been no reactivations to date. The authors express considerable optimism as regards this policy and state that the experience serves somewhat to counterbalance their previous concept that these cavities should be removed surgically or collapsed by thoracoplasty prior to the time when the patients could hope to return to their families and earn a living.

The experience of Bell and associates[15] has been at variance with this. In a comparison of the results of medically treated and surgically treated patients with "open-negative" syndrome, they report a relapse rate of 43 per cent in a group of 118 treated medically and a relapse rate of only 8.4 per cent in 153 surgically treated patients. They conclude that pulmonary resection is valuable in improving the over-all results in patients with "open-negative" syndrome.

Cole and Alley[16] present an analysis of 513 patients of which 15 died within 90 days (2.9 per cent), 10 died late (1.9 per cent), and 8 others died apparently unrelated to surgery or tuberculosis, making a total of 4.8 per cent deaths for surgery and tuberculosis, and a 6.4 per cent over-all mortality. Follow-up over 1 to 5½ years indicated good results in 85.4 per cent.

A report of relapses occurring after resection for pulmonary tuberculosis by Hekking[16] showed a relapse rate of 7.4 per cent in 525 cases of resection observed from 2 to 6 years. The conclusion is reached that the relapse rate has decreased since the introduction of pulmonary resection. The chance of development of relapses after pulmonary resections is increased by resection of cavitary processes, by resection in the active stage, and by resection in patients who have shown a low resistance against tuberculosis.

DISCUSSION

Our attitude toward resection over the years has been a conservative one. In general, it can be stated that our indications consist mainly of cavitary lesions and significant caseous nodular residuals, with or without positive sputum, that persist after 6 to 8 months of chemotherapy.

It is obvious that the extent of resection will depend largely upon the distribution of the lesion and other anatomic and technical considerations. When consistent with these limitations, it is our policy to excise the least amount of lung possible with a view to minimal encroachment upon pulmonary function. In the light of this policy, it can be seen that an inevitable consequence has been a steady decrease in the number of pneumonectomies and a sharp increase in the number of segmental and subsegmental resections. The former now represent less than 2 per cent of all resections and the latter accounted for 43 per cent of all resections in 1957.

Although a rather higher incidence of fistulas was encountered in segmental resection, the mortality was not significant enough in relation to other considerations to alter the trend toward its more frequent use. However,

when the removal of cavitary lesions with positive sputum is contemplated, even though clearing has occurred in the other segments of a lobe, it is thought unwise to perform segmentectomy because of the danger of transection through disease in the intersegmental plane. This applies particularly to the right upper lobe lesions where lobectomy is often preferable.

The majority of moderate-sized residuals with or without cavity, which are reasonably well localized, present no particular problem. It is generally agreed that the indication for resection of these lesions is clear and, indeed, mandatory.

The smaller nodular lesions must be assessed on the basis of such factors as age, sex, previous behavior of the lesion, and prior bacteriologic status rather than any arbitrary lower limit of size. It is felt that the majority of these small lesions represent dangerous sources of reactivation and that prophylactic resection is indicated. The follow-up studies of comparable lesions treated medically are far from conclusive as regards the protection afforded against relapse by long-term chemotherapy.

At the other extreme, in the widely distributed lesions, an aggressive approach is frequently dictated, despite admittedly higher operative hazards, when it seems feasible to remove residual areas of disease which appear to be dangerous foci from the standpoint of the probability of subsequent reactivation. Such lesions can be operated upon with reasonable safety when respiratory reserve permits. When such factors as age, emphysema, poor pulmonary reserve, and general debility combine to increase operative risk significantly, a compromise must be made occasionally in favor of prolonged chemotherapy even in the unequivocal open-negative lesion. When cavity is not definitely discernible by laminagraphy and the above factors obtained, the decision will perhaps even more often be made against resection.

The unequivocal indication for the employment of surgical excision is the persistence of positive sputum in the face of an adequate trial of chemotherapy. It is unfortunate that this group of patients in whom surgery is most urgently needed, suffers the highest complication rate and mortality, regardless of the type of resectional procedure employed. This is strikingly illustrated by the fact that in the 18 per cent of patients in this report, who had positive sputum immediately prior to operation, the mortality rate was 13 per cent, and 13 per cent relapsed. This group of patients with positive sputum was responsible for one half of the operative deaths, one half of the relapses, one third of the bronchopleural fistulas, and two thirds of the spreads that occurred in the entire series.

In view of the almost certain failure of treatment and grave prognosis of this group without surgery, it is felt that these significantly higher morbidity and mortality rates are not excessive and must be accepted and every possible effort made to remove the major residual lesion. The control of remaining widely scattered areas of disease must then be entrusted to whatever available drugs may be most effective.

The successful management of this difficult problem requires the closest collaboration of the surgeon and his medical colleagues. A significant salvage

rate in this group can be achieved only by careful preoperative assessment of cardiopulmonary capacity, the evaluation of the severity of associated degenerative disease, the manipulation of available drugs to best advantage, the skillful application of surgical techniques and the exercise of good surgical judgment.

Our experience demonstrates that the relapse rate following surgery is not great, being 5.0 per cent for the entire group. However, these patients who relapse have done very poorly, since approximately 70 per cent of them are still active or have died. The over-all experience in our clinic with both medical and surgical relapses is that about two thirds of these recover. The disadvantage which obtains in the surgical case is explainable by the exhaustion of medical and surgical resources in attaining the inactive status in the first place. The control of many of these individuals is a tenuous one at best and often was accomplished with patients who had had extensive chronic disease of long duration. Furthermore, they are often elderly and have had a poor response to chemotherapy for a variety of reasons.

Resectional surgery has proved to be a valuable adjunct to the treatment of pulmonary tuberculosis and has become increasingly safe in conjunction with adequate preliminary treatment with the antituberculosis drugs. Its usefulness in a broad central area where the indications are unequivocal is such as to demand its employment. Its value as insurance against relapse is such that prophylactic resection must be seriously considered even in very small residual lesions. A positive, even aggressive, approach to the problem of the group of patients with extensive disease in whom the prognosis is otherwise grave is believed to be indicated if undertaken with due regard to the limitations imposed by lowered pulmonary function, emphysema, and advanced age.

SUMMARY

A review of our experience with pulmonary resection in the treatment of tuberculosis at Herman Kiefer Hospital has been presented. A total of 1,769 resections has been performed upon 1,730 patients. The current status is known in 91.4 per cent of the patients. The last 1,567 resections have been examined in some detail. The operative mortality was 2.9 per cent and the late mortality 1.9 per cent, a total mortality from all causes of 4.8 per cent. There is still active disease in 3.8 per cent.

The incidence of bronchopleural fistula was 6.2 per cent.

Relapse occurred in 83 patients, a total incidence of 5 per cent. Four of these have died, a mortality rate of 4.8 per cent, and 62 per cent still have active disease.

The seriousness of positive sputum at the time of operation is reflected in a significantly higher morbidity and mortality rate. Despite this higher operative hazard, the continued use of resection in this difficult area appears justified.

During the past year, 297 resections have been performed with a total mortality of only 1.3 per cent, or less than half the mortality rate of the entire series.

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(For Discussion, see page 835)

PULMONARY INFARCTION COMPLICATING SEGMENTAL RESECTION

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INTRODUCTION

AT THE PRESENT TIME, the mortality rate following pulmonary resection in the treatment of tuberculosis is very low indeed—an impressive and highly favorable rate never considered possible a few years ago.[1] Such imposing results, however, are not evident when untoward morbidity and complications following resectional therapy are analyzed. The magnitude of these problems is reflected in the voluminous literature, which has appeared since 1946, dealing with postoperative complications.

Early in 1954, one of us (J. M. S.) was impressed by the gross appearance of a subsegment of pulmonary parenchyma, seen at secondary thoracotomy, which had the gross characteristics of infarcted tissue; this subsegment comprised approximately half of the raw surface of the remaining anterior segment of the right upper lobe following apical and posterior segmental resection of tuberculous residuals 10 days previously. Numerous alveolar air leaks, producing a tension pleural space and prompting the secondary thoracotomy, were visualized over the irreparably devitalized subsegment of lung; an intersegmental vein coursing over the infarcted lung was thrombosed without evidence of proximal ligation; incision into the discolored subsegment produced no bleeding. This necrotic lung tissue was easily removed from the underlying normal subsegment, by traction and blunt dissection, down to the subsegmental bronchus which was divided and closed; no appreciable surface air leaks were then evident, after which a limited modified thoracoplasty was performed, with underwater-seal pleural drainage. Subsequent recovery was uneventful. The pathologic diagnosis was "infarction of lung tissue; no evidence of a specific granulomatous process."

Since becoming aware of this problem of localized postresection infarction of the lung, 399 additional segmental resections have been performed at Fitzsimons Army Hospital. Following these procedures, 14 patients, or 3.8 per cent,

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have developed complications, similar to those related above. Apparently, this pathologic entity has not occurred in the experience of many thoracic surgeons or has progressed to the stage of bronchopleural fistula, empyema, or hemorrhage before secondary corrective surgical measures are undertaken.

During this 4-year period, we have become increasingly familiar with the clinical and roentgenographic characteristics of this postoperative complication, and with the method of management we consider proper when such difficulties arise. Suggestions as to prevention will be presented.

REVIEW OF THE LITERATURE

In regard to the surgical anatomy of the pulmonary vasculature, Appleton,[2] in 1944, was impressed with the degree to which segments of the right upper lobe were supplied by trans-segmental arteries. In 22 per cent of 50 cases in his study, the posterior segment was supplied by recurrent branches of the apical segmental arteries. In 8 per cent, the posterior segment was fed in part by branches of the anterior segmental arteries. Similarly, the apical segment received its blood supply partially from rami of the anterior segmental artery. The anterior segment received additional branches from either the apical or the middle lobe arteries in another 8 per cent of specimens. From such findings, it may be concluded that the possibility of impairing the anomalous arterial blood supply to an isolated upper lobe segment is not insignificant. In addition, Appleton was the first anatomist to relate the pulmonary veins to the intersegmental surface and speculate upon their surgical significance.

Unfortunately, the understanding of the pulmonary vasculature is beset by the difficulty of multiple terminology and various investigators have attempted to improve the situation in different ways. Overholt and co-workers[3] suggested that the venous drainage of the bronchopulmonary segment consisted of lobar veins having both segmental and intersegmental tributaries. Boyden[4] felt that this was oversimplification but surgically significant in dissection of the intersegmental plane.

The technique of segmental resection was gradually perfected after the anatomic concept of the bronchopulmonary segment was advanced. In 1939, Churchill and Belsey[5] reported the employment of this technique in the surgical treatment of bronchiectasis, the underlying aim being to preserve as much normal lung tissue as possible. Clagett and Deterling[6] also adapted this technique to the lingula for the treatment of bronchiectasis. The first significant series of segmental resections in the management of pulmonary tuberculosis was reported by Chamberlain and Klopstock,[7] in 1950. They recorded 72 cases so treated and were encouraged to continue its use, noting that there was a decided decline in the number of postoperative complications as their technical skill and postoperative care improved.

Clagett,[6] appreciating the anatomic concept of the bronchopulmonary segment, applied the "traction" dissection technique, beginning at the hilum; a method, with slight modifications, employed by most thoracic surgeons today.

Initially, Clagett infolded the raw surface area, a procedure which apparently led to numerous complications. Overholt left the segmental surface open and allowed the lung to "breathe."[3]

Chamberlain and Klopstock cautioned in regard to the dissection of the left upper lobe hilum noting that there was often an anomalous arterial supply to the anterior segment arising from the parent artery to the apical segment as described by Appleton and Boyden.[2, 4, 7, 8] They pointed out that ligation of such vascular structures, before proper identification, might lead to serious complications. These investigators also clearly visualized the intersegmental veins as receiving tributaries from both sides of the intersegmental plane and advised careful dissection and preservation. Chamberlain and Klopstock also suggested that "it is a good general rule to delay the division of any vein until absolutely necessary."[7]

Most of the experimental work in the pathogenesis of pulmonary infarction has dealt with embolization or ligation of the pulmonary arterial tree. Although infarction of pulmonary tissue may occur in the presence of sudden occlusion of a large pulmonary artery in a healthy animal, it is not the usual course of events.[9, 10] It is well established that any condition leading to the production of serious venous stasis predisposes to subsequent infarction of arterial origin.

Ameuille and co-workers[11] were concerned with the character of collateral circulation developing in the presence of pulmonary venous ligation. Only 4 of 10 dogs survived his intrathoracic procedure and these were sacrificed at intervals up to 7½ months after which injection studies revealed marked vascularization of the overlying pleural plaque, opposite the operative site, forming collateral venous channels. Such a mechanism of venous return is absent in a nonexpanded isolated segment such as occurs in complicated segmental resection. In 1938, Reinhoff,[12] in describing a two-stage technique of pneumonectomy for bronchogenic carcinoma, referred briefly to the danger of inadvertently ligating the superior or inferior pulmonary veins. He suggested this could lead to moist gangrene. He cited, however, no specific cases and did not speculate upon the pathogenesis. Brantigan,[13] and Kent and Blades,[14] in discussing the anatomy of the pulmonary lobes, implied that gangrene might result if the venous drainage of the related lobe was interrupted. In 1948, Swan and Mulligan[15] did experimental ligation of pulmonary veins in dogs. Pathologic study revealed severe fibrinopurulent pleuritis with vascular adhesions, intense hyperemia, hemorrhage and edema of the alveoli and bronchioles, and increased susceptibility to infection. They noted thrombosis of the veins but did not observe extensive tissue necrosis. They concluded that, in spite of the initial congestion and alveolar hemorrhage, the lung eventually recovered with approximately a 20 per cent decrease in functional capacity. Perhaps in these experiments, the rapidly developing vascular pleural plaques bridged the gap preventing acute lobar pulmonary venous insufficiency. Wyatt and co-workers[16] conducted a similar canine study and noted the importance of antibiotics (penicillin) in preventing severe postoperative infection predisposing to infarction. They were impressed with the extent and vascularity of the pleural plaque and commented upon its significance in establishing collateral circulation. These

investigators described small fibrous nodules scattered throughout the healed areas in the pulmonary lobe and speculated upon their pathogenesis without considering them healed infarcts.

In 1951, Stemmermann, Daniels, and Auerbach[17] ascribed the pathogenesis of bronchopleural fistula, following excisional therapy for pulmonary tuberculosis, to pre-existence of disease in the pleural space at the time of surgery. The resultant empyema spread to the bronchial stump with subsequent breakdown and fistulous formation. In 1952, Zelman, Davidson, and Ulmar[18] reviewed 200 cases of pulmonary tuberculosis submitted to surgical resection and found that necrosis of the lingula occurred in 1 case following segmental resection. Steele, Narodick, and Cadden,[19] in 1953, remarked that the disadvantage in the performance of small resections for pulmonary tuberculosis was the high incidence of postoperative complications. Although there was no mortality and the end results were good, 20 per cent of patients developed persistent lung leaks, hemothorax, or empyema. There were, however, no tuberculous complications. In the presence of such leaks, they advocated that it was better to perform a secondary thoracotomy than to persist in the prolonged use of drainage tubes. Bell,[20] in 1955, discussed the postresection space complicating segmental and wedge resection. He reported 61 complications in 308 resections, an incidence of 20 per cent, and noted that the location of the persistent space was usually at the operative site. In his experience, most localized pneumothoraces responded to underwater-seal drainage within 2 weeks; a small number, however, became chronic problems with pyogenic infection. He concluded that the occurrence of such complications did not significantly influence the future status of the patient's tuberculosis. Douglass, Bosworth, Judd, and Chang,[21] as all of us, had some experience with late leaks or pneumothoraces occurring 2 to 10 days after removal of intrapleural tubes. They used the term "alveolo-pleural fistula" and remarked that a distinction between this problem and bronchopleural fistula should be made. Corpe, Shek, and Cope,[22] in 1956, reviewed 182 segmental resections performed for tuberculosis, and noted that air leaks in the postoperative period accounted for more than half of the total number of complications. Twenty-two patients having pleural spaces required further therapy. Of these, 3 developed bronchopleural fistulas, empyema subsequently developing in 1. Nine of the patients were treated by re-insertion of pleural catheters and 12 by postexcisional thoracoplasty. Final results were considered to be excellent; however, they were unable to make recommendations which might decrease the significant postoperative morbidity rate. In 1955, Findlay, while a member of the Thoracic Surgery Staff at Fitzsimons Army Hospital, became interested in our studies on postoperative pleural space problems and was convinced that postresection pulmonary infarction does occur and, not infrequently, is the sole cause of the residual pleural air pocket which may persist and progress to other complications of a more serious nature, if not promptly and properly treated. In 1956, he reported that a persistent pleural space following partial resection of the lung may lead to bronchopleural fistula months later, and advised early reoperation at which time the visceral pleura is not thickened with

fibrosis, and the lung is still expandable.[23] One of us (J. M. S.) reported on some of our early experiences and opinions on "postoperative infarction of the lung" in the Eighth Annual Symposium on Pulmonary Diseases—Fitzsimons Army Hospital, 1955.[24]

CLINICAL FEATURES AND OBSERVATIONS

The complication of pulmonary infarction following segmental resection for pulmonary tuberculosis occurred fifteen times in 400 procedures performed on 371 patients during the period of January, 1954, to February, 1958. This difficulty involved 7 males and 8 females with an average age of 28 years. All except 3 were Caucasian. The average duration of tuberculosis diagnosed prior to surgery was 15.4 months, the longest being 5 years and the shortest 7 months. The period of chemotherapy prior to surgery averaged 7.4 months, the longest course being 15 months, in 3 cases 6 months, and in 1 only 1 month. Chemotherapy primarily consisted of isoniazid, para-aminosalicylic acid and streptomycin in varying combinations. The average duration of negative cultures prior to surgery was 6.8 months, the longest being 12 months and in 1 it was only 3 months. One patient who had never shown positive cultures received 1 month of preoperative chemotherapy.

The surgical description of the adjacent segments remaining at the conclusion of surgery revealed no palpable disease in 8 cases. Five had a definite nodular fibrotic consistency to palpation and 2, upon dissection of the hilum, revealed anomalous vessels. In 9, there was either severe fibrosis obliterating normal tissue planes or nodular disease within the intersegmental plane. In 1 case, a small cavity was opened along the intersegmental plane and this was the patient who exhibited a late tuberculous pseudopulmonary cavity of the type described by Bell and Medlar.[25] Review of the operative reports revealed no technical difficulty in 9 cases although in these patients there was frequent mention of ligation of venous channels and oversewing of leaking segmental surfaces. In the remaining 6 cases, the dissection was described as difficult, requiring sharp procedures and wedging across the intersegmental plane (see Table I).

Pathologically, all specimens obtained at the first procedure revealed cavitary disease except four which consisted, primarily, of healing necrotic foci. Two cavities were considered to be inspissated while three were reported as "healed." In one there was histologic evidence of progressive disease. The tissue removed at the original procedure usually consisted of the apicoposterior segment of the upper lobe, there being seven such procedures on the left and three apical and posterior segmentals on the right. Of the remaining five, two consisted of removal of only the posterior segment of the right upper lobe, the posterior subsegment of the left upper lobe, the apical segment of the right upper lobe and finally a left upper lobe equivalent. When pulmonary infarction complicated the segmental procedure, it always occurred in a segment of which one segmental plane was involved in the dissection procedure. Thus, the anterior segment was the commonest such segment involved, being adjacent and exposed

in ten procedures. The three posterior segmental resections subsequently involved subsegments of the apical segment. The 1 case in which apical resection was performed involved a subsegment of the anterior segment and, finally, following removal of the left upper lobe equivalent, infarction occurred in the lingula.

In all cases, the essential features leading to re-operation for presumptive infarction of an adjacent segment or subsegment consisted of a persistent air space, usually apical or "sub-tentorial" (if pleural tent created), and overlying a triangular density with its broad base directed medially and often having a concave upper margin. This apical air space occasionally showed a fluid level; however, none were successfully collapsed by needle or catheter aspiration. Inordinate pain, persisting beyond the immediate postoperative period, was a noticeable feature in one third of the cases. A sharp tearing pain in the affected region was sometimes reported. These patients as compared with others seemed to bleed more profusely in the early postoperative period. Three had hemothoraces treated by lytic agents (streptokinase-streptodornase) prior to removal of intrapleural tubes after initial thoracotomy.

The time which elapsed between initial segmental resection and re-operation averaged 17.6 days in 14 cases. In half of the patients, the second thoracotomy was performed in 9 to 14 days. The longest interval of 144 days occurred in a patient whose initial postoperative course was considered essentially normal. After being released to the Medical Service, there developed a gradually enlarging apical subtentorial air space with the characteristic underlying triangular density. Upon re-operation, a tuberculous pleuropulmonary cavity was demonstrated; this being the only case in which an active tuberculous lesion was proved at secondary thoracotomy. The subsequent course has been uneventful.

In regard to the first operative procedures, pleural space obliterating procedures were performed in 10 cases of which 8 consisted of construction of a pleural tent and in 2 a modified thoracoplasty was performed.[26] In 3 cases, partial obliteration of the pulmonary space created after segmental removal was accomplished by pleural margin approximation employing nonsuture cotton ligatures in 2, and interrupted sutures in 1.[29] All patients except 1 received intrathoracic instillation of streptomycin and penicillin.

At re-operation, an apical or subtentorial air space was detected of varying dimensions having an average volume of 100 to 300 c.c. The average space diameter was 5 to 6 cm. In all instances, air leaks could be demonstrated over various areas of the infarcted tissue. Upon stripping fibrinous material from the affected segment, the latter characteristically did not bleed but bubbled diffusely and presented a firm, woody, or soft, frankly necrotic texture to palpation. Its color varied from purplish-red to brownish-black depending upon the stage and the extent of the necrosis. In 2 cases, there was a frank odor of gangrenous tissue.

The extent of the infarction was variable. It always involved some portion of the intersegmental surface and characteristically involved the most distal

TABLE I. INFARCTION COMPLICATING SEGMENTAL

CASE NO.	AGE, SEX, AND RACE	ADJ. REMAINING SEGMENT*	INTERSEGMENTAL PLANE†	SURGICAL DIFFICULTY‡
1	24 W. M.	Clear	Nodular disease in intersegmental plane	"No difficulty"
2	35 W. F.	Clear	Well-defined	"Intersegmental veins demonstrated"
3	15 W. F.	Nodular, anomalous vessels	Fibrotic, ill-defined	"Sharp dissection"
4	31 W. M.	Anomalous blood vessels	Well-defined	No technical difficulty
5	35 W. M.	Clear	Clear	Clamping and ligation of veins in intersegmental plane
6	23 W. M.	Clear	Well-defined	"Ligation of 'distal' pulmonary veins of intersegmental plane"
7	25 Japanese F	Clear	Well-defined	"Veins draining intersegmental plane ligated and cut"
8	25 Spanish F	Clear	Fibrotic	Clamping and ligation of veins in intersegmental plane
9	20 W. M.	Nodular	Nodular disease in intersegmental plane	Wedging and ligation of "many" intersegmental veins
10	33 W. F.	Nodular	Fibrotic, ill-defined	Wedging across intersegmental plane
11	24 N. F.	Nodular	Fibrotic	Sharp dissection required; difficult
12	39 W. F.	Clear	Well-defined	"Clamping and ligation of intersegmental vessels"
13	42 W. M.	Clear	Considerable fibrosis at hilum	"Definition of segmental plane accomplished but difficult to start"
14	17 W. F.	Nodular	Fibrotic	"Excessive oversewing of intersegmental vessels"
15	32 W. M.	Nodular	Opened cavity in intersegmental plane	"Wedge across intersegmental plane"

*Description of the adjacent remaining segment in which infarction subsequently occurred.
†Report on the condition of the affected intersegmental plane at the time of the initial surgery.
‡Pertinent difficulties encountered in performance of initial segmental resection.

RESECTION FOR PULMONARY TUBERCULOSIS

SEGMENTS REMOVED§	SPACE OCCLUSION‖	SEGMENT INFARCTED	INTERVAL¶ (DAYS)	APPEARANCE AT RE-OPERATION#
Apical and posterior RUL	None	Anterior subsegment RUL	35	Necrotic blood clot; numerous air leaks
Apical RUL	Pleural approximation anterior segment to posterior segment	Anterior subsegment RUL	12	Intersegmental veins thrombosed; leaks discernible
Posterior RUL	None	Apical RUL	9	Numerous small BP fistulas
Apical and posterior RUL	Pleural tent	Anterior subsegment RUL	16	Demonstrable surface air leaks
Apicoposterior LUL	None	Anterior subsegment LUL	10	Localized surface air leaks
Apicoposterior LUL	Thoracoplasty (modified)	Anterior subsegment LUL	16	No comment by surgeon
Posterior RUL	Pleural tent	Apical subsegment RUL	13	Surface air leaks
LUL equivalent	Pleural tent	Lingula	10	Numerous small BP fistulas
Apicoposterior LUL	Pleural tent	Anterior segment LUL	22	Air leaks; surgical hemorrhage pulmonary artery
Apicoposterior LUL	Pleural tent	Anterior subsegment LUL	10	No comment by surgeon
Posterior LUL	Pleural approximation anterior segment to superior segment LUL	Apical LUL	10	Localized surface air leaks
Apical and posterior RUL	Thoracoplasty (modified)	Anterior segment RUL	14	Foul ordor; visible sutures; hemorrhage from pulmonary artery
Apicoposterior LUL	Pleural tent	Anterior segment LUL	29	Gangrenous; visible thrombosed vessels
Apicoposterior LUL	Pleural tent	Anterior subsegment LUL	41	Surface air leaks over infarcted area
Apicoposterior LUL	Pleural tent, anterior segment to superior segment LLL-pleural edges	Anterior subsegment LUL	144	Many sutures in necrotic tissues; 3 air leaks (minute)

§Bronchopulmonary segments removed during the first thoracotomy.
‖Concomitant space obliterating procedure performed at the time of initial segmental resection.
¶Time which elapsed between the initial segmental resection and re-operation for infarction.
#Appearance of the intersegmental surface at the time of re-operation.

portion of the specimen; sectioning revealed extension of varying distances into the underlying parenchyma. In 3 cases there was complete infarction of the entire related segment. When surgery was performed under 3 weeks, there was no great technical difficulty encountered in removing the infarcted segment or subsegment; however, there was an exception in 1 case as related below. A fairly clear line of demarcation was usually visible between necrotic and viable lung. Often peripherally the necrotic tissue could be wiped away with a gauze sponge or separated by blunt dissection. The main difficulty in the dissection was encountered at the hilum where, because of close proximity of the pulmonary vessels and the severity of the necrotizing process, serious hemorrhage may ensue, due in 1 of our cases to involvement of the wall of the pulmonary artery by the necrotic process. However, the outcome was entirely successful.

Follow-up studies for periods, ranging from 3 months to 4 years, have revealed no evidence of spread or reactivation in any, and no complications attributable to the second thoracotomy. Additional data pertaining to the 15 patients who had localized postresection infarction of the lung are listed in Table I.

PATHOLOGY

Grossly, the specimens were purplish-black to brownish in color and had a variable consistency. Most presented a firm, indurated, noncrepitant texture but, in some cases, the material was markedly soft and friable. Thrombosis of surface veins buried in blood clot or fibrin along the intersegmental plane was frequently observed. The pleural surface of each specimen was often partially covered by a fibrinous layer having a variable consistency depending upon the time which had elapsed since the previous surgery. This layer was usually soft, friable, and yellowish-white in color. The superficial surface of this plaque was often firmer than the underlying surface where it was in apposition with the necrotic tissue.

The cut surface of specimens usually revealed a dry, finely granular, purplish-black appearance which was sharply demarcated from any comparatively normal lung that may have been included with the specimen. In two specimens in which there was a distinct odor of gangrenous tissue, central softening and liquefaction were observed. Examination of the cut intersegmental surface often revealed thrombosed vascular structures leading into the necrotic lung.

Microscopic examination of sections taken at right angles to the segmental surface revealed characteristic hemorrhagic infarction with focal necrosis of pulmonary tissue tending to become confluent at the surface. The central portions of these lesions showed complete loss of cytologic detail including the nuclei of alveoloseptal cells. In spite of such profound necrosis, the tissue characteristically maintained its configuration with well-defined acidophilic hyaline walls inclosing degenerating masses of conglutinated red blood cells. Although occasionally the periphery of the infarcted area was well circumscribed by fibrous tissue (probably representing a sublobular fibrous tissue plane), it was more characteristic to find no such delimitation, the zone between

viable and nonviable tissue being remarkably free of either fibroblastic tissue or inflammatory cells. Healing was evidenced by a feeble and ineffective organization of intra-alveolar fibrin resulting in thick-walled avascular nonfunctional alveoli.

The contained vessels showed thromboses and mural necrosis, particularly noticeable in the superficial intersegmental veins. Serial sections of the latter revealed proximal viability and luminal patency. Spaces within necrotic tissue were often observed; this was felt to be histologic representation of the multiple alveolopleural fistulas so characteristic of these lesions.

Three aspects of this material appeared particularly interesting to one of us (H. N. H.). Elastic stains upon necrotic tissue showed a surprisingly intact architecture despite the profound necrosis, and the prolonged interval following initial surgery. The necrotic tissue tended to become hyalinized and except for 2 cases there was little evidence of actual lysis of tissue. The second puzzling feature of these specimens consisted of a marked degree of endothelial proliferation and, often, luminal obliteration of related arterial structures. These were often observed in adjacent viable pulmonary tissue. The endothelial proliferation might be interpreted on the basis of adjacent tuberculous disease, however, the presence of an organizing thrombus would tend to discount this possibility. The final impressive feature was the degree to which metaplastic bronchial squamous epithelium was disrupted and dispersed among the peripheral layers of necrotic tissue.

BACTERIOLOGY

It is not surprising that infection did occur in 3 of the 15 cases. Such infections were confined to the infarcted segment, subsegment, or pleuropulmonary cavity. In 2 cases (12, 13; see Table I) there was frank gangrenous tissue, due presumably to a Vincent-type infection, although cultures were sterile. The final case (15) was the only 1 showing evidence of an active tuberculous process in tissue removed at the second thoracotomy. The patient had a prolonged delay of 144 days before secondary thoracotomy and acid-fast bacilli were demonstrated on smear and tissue stains in the wall of the cavity; however, cultures were negative. In this instance, there was formation of a tuberculous pleuropulmonary cavity.

CASE REPORTS

Three clinical cases which illustrate this postresection syndrome of localized pulmonary infarction are presented below. The other 12 cases were rather uniformly similar to those here recorded; thus, an accurate preoperative diagnosis was made in our last 8 patients.

CASE 2.—A 35-year-old white woman was admitted on April 9, 1954, with a diagnosis of pulmonary tuberculosis. Numerous roentgenograms, since 1951, revealed a nodular infiltrate confined to the area of the right upper lobe which was considered to have undergone no change. However, in February, 1954, two routine gastric cultures were positive for acid-fast bacilli. Other laboratory studies and physical examination revealed nothing remarkable other than post-tussic râles over the right upper chest. Chemotherapy consisted of streptomycin

and isoniazid. On Oct. 22, 1954, after 6 months of chemotherapy, a right upper lobe apical segmental resection was performed for significant residual nodular disease restricted to this segment. The created raw surfaces of the anterior and posterior segments were approximated at the pleural margin by four interrupted sutures. Twelve days after initial surgery, a thoracotomy was again performed because of a persistent air-fluid space which had not responded to multiple thoracenteses (Fig. 1). The midpectoral subsegment of the anterior segment appeared to be infarcted, having a dense, rubbery consistency, and the vessels along the segmental plane were thrombosed; intermittently, air bubbled from this surface. The involved subsegment was removed without difficulty after which the raw segmental surfaces

Fig. 1.

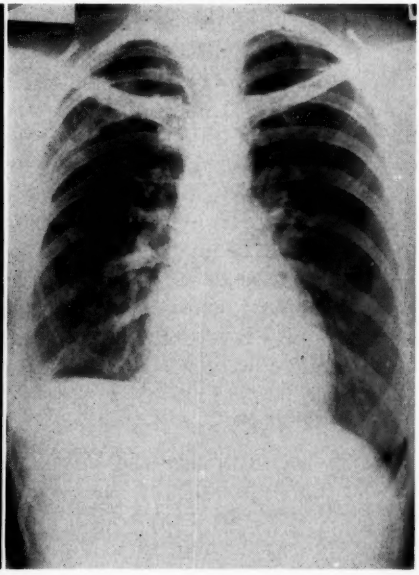

Fig. 2.

Fig. 1.—Right lateral roentgenogram of chest of Case 2 showing postoperative air-fluid space at site of apical segmental resection. Triangular density anterior to space proved to be infarction of subsegment of the anterior segment.

Fig. 2.—Roentgenogram of chest 20 months later. Regenerated fifth rib on right. Patient asymptomatic.

came into favorable apposition as the lung was expanded by the anesthesiologist; the pulmonary remnant filled the hemithoracic space completely. Two large tube pleural drains were connected to water-seal bottles and suction instituted. Subsequent recovery was uneventful and the patient has continued to do well (Fig. 2).

Pathologic examination of the first specimen revealed caseonodose tuberculosis. The second specimen consisted of organizing hemorrhagic infarction of the anterior subsegment with nonspecific chronic pleuritis and pulmonary fibrosis.

CASE 5.—This 35-year-old white man was admitted on April 18, 1956, because of an expanding, rather circumscribed lesion in the left upper lobe (Fig. 3). Skin tests: PPD No. 1, four plus; histoplasmin and coccidioidin, negative. No positive smears or cultures for acid-fast bacilli were obtained. Streptomycin and isoniazid were started on May 18, 1956, and apicoposterior resection was performed June 8. The solitary lesion, 2.2 cm. in diameter, proved to be a rather thick-walled tuberculous cavity from which acid-fast bacilli were isolated.

No concomitant collapse procedure was performed. In the postoperative period, the water-seal suction bottles continued to bubble and an apical air space failed to improve (Fig. 4). He continued to complain of rather severe chest pain. Ten days after segmental resection, he was returned to surgery and an infarction of a subsegment of the anterior segment, from which air leaked freely, was demonstrated and removed. A modified thoracoplasty was performed and the patient has done well since.

CASE 13.—A 42-year-old white man noticed the onset of fatigue 2 years prior to admission. A chest roentgenogram on July 31, 1956, revealed bilateral upper lobe cavitation. The only early positive culture was obtained as the result of bronchial washings. Chemotherapy was instituted on Aug. 8, 1956, with 600 mg. of isoniazid and para-aminosalicylic acid, 12 grams daily. On December 17, the isoniazid was increased to 900 mg. On March 15, 1957, a right thoracotomy was performed with removal of the apical and posterior segments. A tent of the apical parietal pleura was performed and recovery was uneventful. On

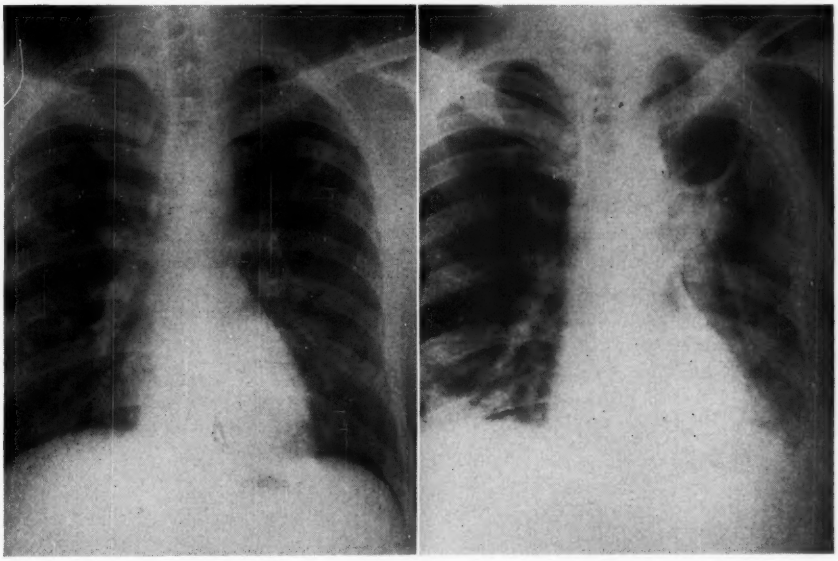

Fig. 3. Fig. 4.

Fig. 3.—Roentgenogram of chest, Case 5, prior to resection of solitary tuberculous lesion in apicoposterior segment.

Fig. 4.—Roentgenogram 8 days after segmental resection. The large tension apical pleural space is demonstrated. Infarction in anterior segment produced the triangular dense shadow below the space.

March 28, 1957, sputum culture was again positive for acid-fast bacilli and the isoniazid was increased to 1,500 mg. with 1 gram of streptomycin daily. On July 10, 1957, an apicoposterior resection of the left upper lobe was performed. Postoperative x-ray studies revealed well-expanded lung with some cloudiness over the apex without discernible space. On July 14, the intercostal tubes were removed. However, on July 26, 1957 (Fig. 5), an air space was observed at the apex and it was felt that this had resulted from an infarcted segment. Air leak was continuous as demonstrated by needle aspiration. He complained of almost constant "sharp tearing" chest pain. Twenty-nine days following the initial left thoracotomy, the left thoracic cavity was entered through the previous operative site; pleural symphysis was observed over an extensive area, and incomplete infarction of the anterior segment was seen

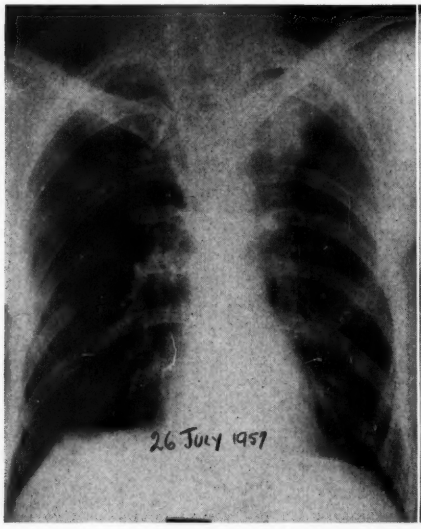

Fig. 5.

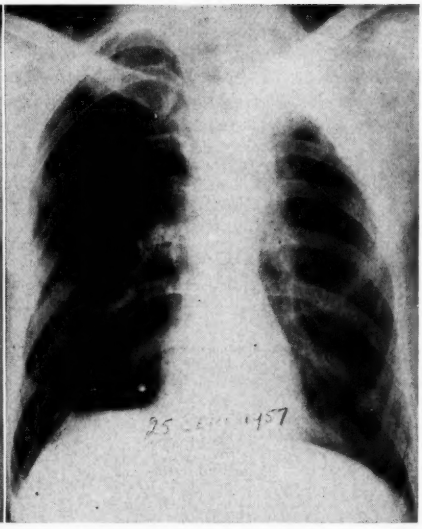

Fig. 6.

Fig. 5.—Roentgenogram of Patient 13 taken 16 days after apicoposterior resection on the left where dense, roughly triangular shadow is demonstrated below apical space. Good contralateral expansion is noted 4½ months after apical and posterior segmental resection and utilization of a pleural tent on right.

Fig. 6.—Roentgenogram of chest 2½ months after second thoracotomy and concomitant upper stage modified thoracoplasty, left, in management of postresection infarction in the anterior segment.

Fig. 7.—Microscopic appearance of hemorrhagic pulmonary infarction (Patient 13), showing characteristic focal necrosis, air spaces, and vascular thromboses. Compressed alveoli and poor delineation left lower area.

which had advanced to the stage of gangrene in one area. Small air leaks on the surface of this infarcted segment were observed and thrombosed vessels leading to the anterior segment were palpated and visualized. Segmental resection of the infarcted segment was accomplished and a modified thoracoplasty was performed. Subsequent course has been completely uncomplicated (Fig. 6). The pathologic specimen consisted of the anterior segment showing a large area of infarction (Fig. 7).

DISCUSSION

Pulmonary infarction complicating segmental resection, according to our concepts, is certainly not a new entity. The characteristic symptomatology has long been recognized, but the underlying pathogensis has not. Such terms as the "late space problem," "alveolo-pleural fistula,"[21] "masked broncho-pleural fistula,"[27] "occult broncho-pleural fistula"[21] have been offered in an effort to characterize this problem or its complications. The salient clinical findings and basic diagnostic features are: (1) a persistent tension air space in the region of the previous pulmonary resection, (2) no evidence of an overt bronchopleural fistula, and (3) commonly demonstrated roentgenographically, below the pleural space, is a triangular density with its broad base directed medially and having a concave upper margin if no fluid is present, otherwise, a horizontal air-fluid level.

The complications which may accompany or follow such pulmonary infarctions, if not corrected by early and proper surgical measures, are as follows: (1) A persistent and nonresolving air space, provided the resulting pulmonary infarction is of significant depth and magnitude. Such may negate the intended purpose of segmental resection, i.e., the preservation of uninvolved and apparently normal functioning pulmonary segments; thus, the space plus the fixed necrotizing and/or organizing segment will deter adjacent segments from expanding to full functional capacity. (2) Bacterial infections—blood, serum, avascular tissue, and the "dead space" provide an excellent culture media for bacterial growth; the intermittent ingress of respired air via the porous necrotic segmental surface is a factor worthy of mention. With due deference to antimicrobial agents, nevertheless, we must assume that, in due course of time, some untoward superimposed complication of a bacterial nature will ensue. A listing of such possibilities is proposed as follows: localized empyema, mixed empyema (pyogenic and tuberculous), tuberculous pseudopulmonary cavity, adjacent specific or nonspecific pneumonitis, contiguous tuberculous reactivation and/or distant spread, putrid lung abscess (such has been observed in our experience on one occasion, however, the exact nature of the pathogenesis was not determined), osteomyelitis of ribs, and chest wall infection. (3) Frank and indisputable bronchopleural fistula. (4) Massive fatal pulmonary hemorrhage—such has been reported in the literature following segmental and subsegmental resection.

The prevention of this type of postoperative complication, i.e., localized pulmonary infarction, depends to a large degree upon maintaining the integrity and continued patency of the remaining branches of the intersegmental vein. It is evident, when considering Column 3 data in Table I (Surgical Difficulty), that the crux of the problem appears to be the surgically exposed surface venous

channels, which, no doubt, play an important role during the immediate postoperative healing phase. It is noteworthy, that the surgeons reported various types and degrees of difficulties in 12 of the 15 patients during dissection in the intersegmental plane. Thus, many factors influence the postoperative competence of the intersegmental pulmonary veins. Some factors which immediately come to mind are: active or fibrotic tuberculous residuals involving the intersegmental plane; tuberculous nodules beyond the intersegmental boundary which are removed by additional wedge excision; excessive bleeding from intersegmental plane, requiring clamping, ligatures and sutures; excessive air leaks, the control of which may also require clamping, ligatures and sutures with possible inclusion of venous tributaries; rare anomalous venous distributions; and technical errors and difficulties. It might be noted that nine surgeons performed the initial surgery on the 15 patients developing this complication; two being staff surgeons and seven were residents on the Thoracic Surgery Service—all being capable of operating independently or had staff surgeon assistance and supervision.

An additional factor which seems to warrant consideration is the possible prophylactic value of the surgically created pleural tent. We feel such a procedure may be of some value if the pulmonary surgical defect is relatively flat (not a deep "wedge like" defect); thus, the pleural tent seems more commonly indicated after an upper lobectomy than following segmental resection. The prevention of this postoperative complication may depend also upon the degree of early angioplasia and venous channels which form in the fibrinous plaque separating the remaining segment from the adjacent pleural space. If there is rapid organization of this surface plaque, then minimally infarcted zones will probably be effectively walled off and healing will take place. On the other hand, if early angioplasia and fibroblastic proliferation is poor, the necrotic surface will continue to leak via multiple alveolopleural fistulas.

This early fibrinous plaque covering the raw surface is uniformly demonstrated in the experimental animal as well as in patients having had pulmonary resections and is a major contributing reparative process which makes the technique of segmental resection possible. This consideration should always be kept in mind by the surgeon who, in some instances, may elect to enhance this aspect of the surface healing process by performing a pleural tent or a limited modified upper-stage thoracoplasty. We have shown to our satisfaction on numerous occasions, after performing pulmonary dissection techniques on dogs, that the rapidly forming fibrinous pleural plaques make it extremely difficult to produce alveolar air leaks of sufficient duration to produce pleural air spaces, although we rather uniformly establish areas of subsegmental infarction by ligation of intersegmental veins.[28]

CONCLUSIONS AND RECOMMENDATIONS

It seems superfluous to state that surgical skill, judgment, and experience are all important, nevertheless we are convinced that any thoracic surgeon,

regardless of experience and training, may from time to time be confronted with this postoperative complication. Therefore, it seems imperative that all should keep this syndrome in mind when postresection space problems occur. It is very questionable if it is proper to apply conservative methods of management such as multiple thoracenteses and re-insertion of catheters and tubes for lengthy periods. Most of our patients have been given the benefit of such methods of early management without any evidence of success. Accordingly, it is proposed that if such methods fail in 4 to 6 days that secondary thoracotomy is in order. Re-inserted closed drainage tubes should not remain long enough to initiate or contribute to secondary infection. It is our opinion that secondary thoracoplasty alone would be curative in only a small percentage of cases; however, such a procedure of the modified type or construction of an apical parietal pleural tent concomitantly with secondary thoracotomy will not infrequently be necessary.

At this juncture of our incomplete understanding of this problem of postsegmental infarction of the lung, we hesitate to make any concrete or dogmatic recommendations and proposals other than those related above. Nevertheless, a "scheme of surgical conduct" is offered which we believe is sound in principle as based upon our clinical experience and incompleted laboratory investigation of this problem to date.[28]

Recommended "Scheme of Surgical Conduct."—

1. Maintain integrity of branches of intersegmental vein on surface of retained segment. Segmental surface clamping, ligatures, and stitch sutures are kept to a minimum; hemostasis is often surprisingly complete after complete expansion of the segment by the anesthesiologist; meticulous and gentle dissection in the intersegmental plane is mandatory.

2. Do not attempt suture infolding of raw segmental surface.

3. Do not attempt reconstitution of pulmonary remnant by multiple layer sutures beginning in the depth of the postresection defect; such closure technique would seem to contribute to intersegmental venous stasis, thrombosis, and subsequent infarction.

4. Complete removal of segment peripherally is essential; dissection may easily progress outside the intersegmental plane, leaving a "nubbin" of subsegment quite devoid of circulation, although expansion may be surprisingly good.

5. Perform upper lobectomy on the right in favor of apical and posterior segmental resections planned, i.e., if fibrosis and nodular disease are found to involve the apical and anterior intersegmental plane. Pleural tent or limited modified thoracoplasty may seem indicated.

6. A similar situation on the left, as related in 5 above, with apicoposterior resection contemplated—if intersegmental fibrosis not overly extensive—suggest that anterior segment be retained, and manage infarction of anterior segment by secondary thoracotomy if such should develop.

7. Secondary thoracotomy in 10 to 14 days when there is clinical and roentgenographic evidence of postoperative pulmonary infarction. Surgical judgment will dictate if concomitant pleural tent or modified thoracoplasty should be performed.

SUMMARY

A series of 15 patients, having incurred pulmonary subsegmental or segmental infarction following segmental resection for tuberculosis in 400 consecutive procedures, has been analyzed. Clinical and roentgenographic features characterizing this postoperative complication are presented. The proposed pathogenesis of demonstrated infarcted contiguous segments and pathologic findings are discussed, and recommendations are offered in regard to prevention. Once frank pulmonary infarction occurs, secondary thoracotomy in 10 to 14 days is recommended. All 15 patients having had secondary thoracotomy are without evidence of reactivation and doing well 3 months to 4 years after segmental resection. The mortality to date in this series of 400 pulmonary segmental resections is 0.5 per cent; 1 patient having died of cardiac arrest at completion of pulmonary resection and the other of pulmonary hemorrhage 6 weeks after upper lobectomy and superior segmental resection.

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DISCUSSION

(PAPERS BY BARRETT [PAGE 803] AND SALYER [PAGE 818] AND THEIR ASSOCIATES)

DR. ROBERT KLOPSTOCK, Brooklyn, N. Y.—The Veterans Administration and Armed Forces Study Group is reporting experiences, year after year, in series of even larger figures. But there are just as many cases of resection performed as the definitive form of therapy for pulmonary tuberculosis, which are not reported. These significant figures themselves represent the efficacy of the resectional therapy for pulmonary tuberculosis.

The majority of thoracic surgeons of the younger generation hardly know any other type of surgical management of pulmonary tuberculosis except resection. The young thoracic surgeon is so exceptionally well trained in pulmonary surgery today because tuberculosis of the lung can be treated by surgical excision with a high degree of safety and thus yield a great volume of suitable surgical cases.

As this Association meets in Boston this year it is perhaps proper to remind the younger generation of chest surgeons present that both the intellectual and surgical conception of the primary elective and limited resection as the surgical therapy of pulmonary tuberculosis emanated from this city's Massachusetts General Hospital. Dr. Churchill and Dr. Belsey designated the pulmonary segment as the surgical unit of the lung in 1939.[1] At the Boston meeting of the American College of Surgeons in 1941, this hospital displayed an exhibit[2] on the surgical pathology of the lung demostrating that the distribution of the chronic suppurative diseases of the lung were restricted to the bronchopulmonary unit with characteristic segmental predilection for each disease, including tuberculosis. In 1943, 15 years ago this month, another study entitled "Lobectomy for Pulmonary Tuberculosis,"[3] proposed the primary and elective resection of the stabilized and irreversible necrotic pathology produced by tuberculosis process in the lung. In this report, the cases of 6 consecutive patients successfully operated upon were presented, exhibiting the variety of the patterns and stages in which tuberculosis of the lung is manifested in man. Opposition to this proposal was raised and it is recorded.

The introduction and general use of specific antimicrobials in the treatment of pulmonary tuberculosis gave us the pharmacologic tool to localize and reduce the tuberculous process in the lung to its irreversible necrotic component within a short time and almost at will. This was a tremendous achievement, but it could be exploited and secured only because the conception and the necessary refined surgical methods for the primary and selective resection of the residual irreversible and potentially dangerous lesions were already at

hand, thanks to the scientific vision of Edward Churchill of this city, at the Massachusetts General Hospital. The rest of the story is being carried out by thoracic surgeons all over the world, among whom is the eminent essayist to whom we have just listened.

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DR. WATTS R. WEBB, Jackson Miss.—I enjoyed very much the discussion given by Dr. Barrett on the trends in the therapy of tuberculosis. In reviewing our series in the Mississippi State Sanatorium, we have noted definite trends, one of which is parallel, and some a little divergent. The first is that of the aging population. During the past 5 years, the average age of our surgical patients has more than doubled, and it would be even greater than that except for the maximum upper age limit beyond which one cannot be operated upon.

The second has been the factor of increasing bacterial resistance. Over a fourth of our patients operated upon fall into the resistant group. In the patient with open-positive lesions and resistant bacilli, the factors keeping these cavities open and allowing the development of resistance appear to be mechanical—bronchiectasis, bronchostenosis, extreme fibrosis, and enlarged nodes around the hilus of the lung. These have proved to require more extensive resections and although, with the use of ancillary drugs, we feel that the short-term results have been good, we feel that the long-term results are going to be poorer. On the other hand, we have not been able to reduce the extent of our resections very much, and still have to do about 20 per cent pneumonectomies.

(slide) This demonstrates the type of lesion, the totally destroyed lung, for which we are doing pneumonectomies. Nothing less than pneumonectomy will suffice in such cases.

(slide) Thoracoplasties, particularly of the plombage type, remain a very excellent modality of therapy in many patients.

DR. JOHN W. BELL, Seattle, Wash.—I would like to compliment Dr. O'Brien's group on a fine review, and their survival statistics are excellent. However, I believe they are somewhat distressed, as all of us are, with the high incidence of bronchopleural fistulas and space problems after segmental resections. One technique that we developed several years ago was that of reconstitution of the lung. We believe that this technique has contributed greatly to the reduction of bronchopleural fistulas and space problems following segmental resection. Our incidence of 20 per cent dropped immediately to 1 per cent and now, in an experience of more than 200 cases, we have had almost no bronchopleural fistulas and no important space problems. We have had occasional bleeding into the bronchial tree with transient hemoptysis. We have never seen pulmonary infarction as a result of reconstitution.

Several details of the technique are important. The sutures should be placed in a vertical manner parallel to the bronchovascular structures rather than transversely across the vascular tree. It is important to begin the reconstitution at the base of the lung at the level of the segmental bronchus and to re-appose healthy lung tissue over the bronchus. We think this goes a long way in avoiding subsequent fistulas. We do not believe that segmental resections should be used in patients with open-positive disease, and still feel that lobectomy is the preferred operation in the uncontrolled case with resistant organisms.

DR. NORMAN WILSON, Boston, Mass.—I too would like to compliment Dr. O'Brien and his group for their analysis of such a large group of cases and for coming up with

so many answers for us. I arise merely to pose some questions in view of the trends today in the surgery of tuberculosis. Those of you who deal with this subject know that we have reached the stage where we have returned to the era of laudable pus and an open cavity being acceptable in the treatment of tuberculosis, and it is very perplexing to see us return to that era without more evidence than we now have. I notice that 18 per cent of their surgical patients had a positive sputum. I would like to ask Dr. Barrett if this was not a more important factor in all the complications than was age and type of operaton, because it has been our experience that the type of operation had absolutely nothing to do with the prognosis of the tuberculous lesion provided you have everything else equal on the board. Pneumonectomy with a good contralateral lung and a clean operation, in a patient still sensitive to the drugs with long effective chemotherapy following surgery, in our experience does as well as does a segmental resection. Most pneumonectomy patients do not fall into this category. I would like to ask if the extent of disease and bacteriologic status of the patient at the time of surgery were not more important problems than were the age and other factors. Also, I would like to ask whether the exacerbation rate was not also related to the same problems because, in our experience, the definitiveness of the operation and the protection postoperatively by effective chemotherapy, not preoperatively, are the factors which keep our patients well.

A third question concerns the positive-sputum group. I believe Dr. Barrett will tell us that these patients were positive in spite of long chemotherapy. In other words, these were not patients who were positive and under active, effective chemotherapy. I would assume they might be those who were clinical failures and had remained positive after many months of therapy, and therefore had gone over the hump of protection and developed increased resistance to the antimicrobial agents.

DR. DAY (Closing).—We want to thank all the discussers for their comments. Most of them were, I would say, favorable. First of all I would like to indicate that we are well aware of the pioneer work of Dr. Churchill and Dr. Klopstock, and we are greatly indebted to them, of course. We are in essential agreement with the statements of Dr. Wilson. We had no opportunity, really, in a large series of this kind, to cover all the points. Of course we agree and tried to point out the fact that positive sputum, at the time of or just prior to surgery, was perhaps the most important common denominator of all our difficulties. We merely aimed to point out that this factor, together with resistance, advanced age, emphysema, etc., further compromised our results.

Dr. Bell's comments I am especially glad to hear, because we too try to reconstitute our segmental surfaces as much as possible when they are located in such a fashion that this can be done; we have not seen any of the dire consequences that are alleged to ensue from such a course of action. Neither have we seen, if I may discuss the last paper at this time too, the phenomenon of infarction to occur as it was said to occur in that series. Perhaps it occurred and we did not recognize it. If one leaves it alone long enough, the patients usually get well.

I think that covers as best I can the various questions asked. I might say that our attitude toward resection in general, as you can see, has been a conservative one over the years and has not changed appreciably except for the tendency to do fewer pneumonectomies and more segmental and subsegmental resections. As Dr. Overholt once said when he passed through our place, the errors that are made in resection are usually made as a result of doing less than the lobar unit. If there is any question of difficulty to ensue from transecting disease in the intersegmental plane, the lobar unit should be removed. Our main indication over the years, of course, has been the open cavity or significant residual following a long period of what we believe to be adequate therapy.

It is our feeling that the analysis presented in this report supports the view that resectional therapy is a procedure of proved value and that its continued use is indicated. In certain areas, the indications are imperative. In the group with small areas of disease, the problem of prophylactic resection is still a controversial subject. At the other extreme,

in the widely distributed lesions, a compromise with regard to the open-negative lesion sometimes must be accepted, when such factors as age, emphysema, and poor pulmonary reserve combine to increase operative risk significantly.

DR. SALYER (Closing).—I wish to thank the discussers. I should add that this postoperative problem, pulmonary infarction of a subsegment or segment, occurs not only at Fitzsimons, but occasionally at other hospitals where pulmonary resection for tuberculosis is done. Presently, we have two ladies in the hospital who had pulmonary resections elsewhere; they now have pleuropulmonary cavitary lesions at the site of segmental removal and highly resistant organisms. After reviewing serial films, we are certain that they developed this specific complication after resection many months ago.

Secondary thoracotomy has been necessary during the past 2 years in 4 additional patients having similar complications following initial resections elsewhere; varying degrees of drug sensitivity of the acid-fast bacilli harbored in these lesions were demonstrated. Presently, these patents are doing well.

Perhaps I should not take issue with Dr. Bell's segmental reconstitution technique since I have never used it; however, when listing all the possible etiological factors and mechanisms which may be related to this postoperative complication, we considered the suture approximation of segmental surfaces as possibly contributing to intersegmental venous obstruction and thrombosis.

Two thoracic surgeons formerly assigned to our hospital have employed Dr. Bell's suture approximation technique; initially they seemed to get good results, later they found that hematomas were prone to develop in the approximated intersegmental plane which would separate and result in an air-fluid space. Such patients were subsequently treated by appropriate surgical methods. It is quite possible that they did not perform the procedure as Dr. Bell does it.

SEVEN YEARS' EXPERIENCE WITH TRANSVENTRICULAR AORTIC COMMISSUROTOMY

Robert P. Glover, M.D., and Howard L. Gadboys, M.D.,** Philadelphia, Pa.*

THE PROBLEMS associated with the surgical alleviation of aortic stenosis have to date presented far greater difficulties than any of the other stenotic valvular lesions. That this is true has been recognized for many years, since the first surgical attempt to relieve an obstructed aortic valve was made by Tuffier in 1913. The exact nature of these difficulties, however, has been fully appreciated only within the past decade, during the recent renaissance of intracardiac valvular surgery. Although as much, if not more, time and valiant effort (both experimental and clinical) have been directed toward the relief of this most intractable of valvular lesions, as compared to tricuspid, pulmonary, and mitral stenosis, equal success has not as yet been forthcoming. There are a number of reasons for this state of affairs.

PATHOPHYSIOLOGIC CHANGES

1. Anatomically, the valve is the least accessible to exploration, for it lies centrally placed within the confines of the heart. The left ventricular myocardium is invariably hypertrophied to a considerable degree. This finding, together with the tremendous vascular pressure generated within its outflow tract, prevents the use of an immediately subaortic transmyocardial incision for the insertion of the finger or instrument, similar to that used in pulmonary valvotomy, because uncontrollable hemorrhage will surely follow. The valve cannot be effectively reached through an auricular appendage. The position of the ostia of the coronary vessels, lying directly behind the valve leaflets, makes direct reconstructive surgery hazardous. An approach from above, directly through the aortic wall, presents the same difficulties encountered in the immediate subaortic transmyocardial approach and results in a surprising incidence of septicemia. Thus, definitive technical approach to the valve is unfavorable.

2. Pathologically, there is an early fusion of the three valve cusps, the margins of which become thickened, rolled, eburnated, and infiltrated with calcium very early in the course of the disease. This is due in part to the

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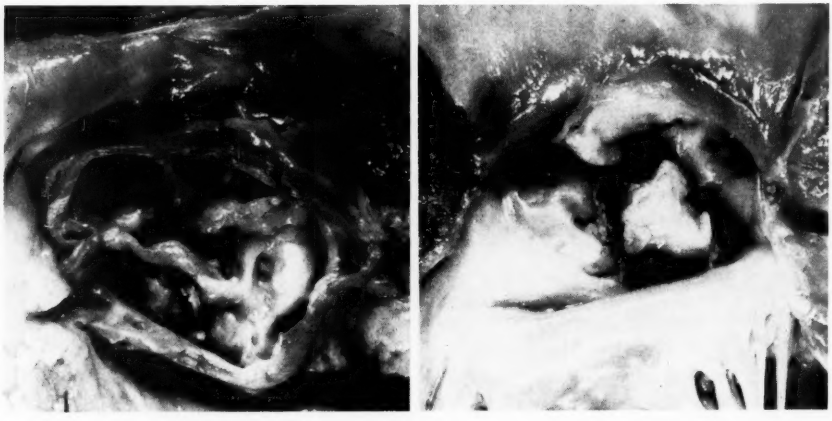
underlying disease itself but also, and perhaps more particularly, to the location of this valve in the course of the bloodstream. The valve lies in a confined space of small diameter and is the recipient of the full head of vascular pressure from the strongest of the contracting cardiac chambers, the left ventricle. The factor of vascular trauma is, therefore, of great importance for, following initial fibrotic agglutination of the cusp margins, there is a constant and steady deposition of traumatic elements, such as fibrin and calcium from which there can be no relief (Fig. 1). Thus, calcification develops early and, as the leaflets are relatively small in surface area, almost complete immobility and fixation may rapidly ensue. Some flexibility of the cusps at their attachment to the aortic ring may remain for a time, but this is less prominent than that seen in the stenotic mitral valve. This factor of trauma no doubt partially explains the



A. *B.*

Fig. 1.—*A*, The aortic valve viewed from the aorta showing the extremes of induration and calcification resulting in complete fixation. *B*, The same valve from the ventricular aspect.

fact that the anteromedial leaflet of the mitral valve, also called the aortic or septal leaflet, is the seat of the greatest calcification in mitral stenosis, for it lies nearest the aortic ring in the high flow-pressure area of the left ventricular outflow tract, and also receives the brunt of vascular ejection at the time of left ventricular contraction. Therefore, from a pathologic standpoint, surgical intervention in aortic stenosis, to restore effectively a measure of normal motion of the valve leaflet, must be carried out before extremes of the above-described process are reached. To wait longer merely compounds the likelihood of surgical failure for, following the valvular change, the left ventricular myocardium hypertrophies, dilates, and in due time wears out and becomes incapable of effective action. As the left ventricle is the cornerstone of the heart and circulation, its failure predicates irremediable diaster. This, again, is in contrast to the findings in mitral stenosis, where, despite profound pathologic and clinical changes, even to the point of prolonged congestive failure and invalidism, the left ventricle retains its indispensable function—actual, compensatory, and recuperative—almost to the end.

3. Physiologically, because of the jetlike ejection of relatively small amounts of blood with each left ventricular contraction, the systolic blood pressure may not reach high levels although it is perhaps sustained and maintained longer than usual. Diastolic pressures, however, remain high so that the pulse pressure is small and coronary artery filling, now under greater stress because of the demands of an increased myocardial mass, may be inadequate. As such, these patients are often precarious anesthetic risks, to say nothing of the superimposed danger of surgery.

CLINICAL CORRELATION OF PATHOPHYSIOLOGIC CHANGES

To recapitulate, therefore, as a result of these pathophysiologic changes, the ultimate prognosis in a given case of aortic stenosis, almost invariably calcified and immobile, lies purely in the integrity of the left ventricular myocardium. This chamber attempts valiantly to maintain adequate systemic circulation. Myocardial work is required to impart velocity to a necessary volume of blood. As the valve area diminishes, increased propulsive pressures accompanying left ventricular hypertrophy are clinically manifested by forceful heart action and heart consciousness. Added demands for increased cardiac output are thwarted by the unyielding valve, and fatigue and palpitation develop. The left ventricular pressure rises in an attempt to overcome the obstruction; a systolic pressure of 400 mm. Hg represents the extreme in our experience. Once a critical valve area is reached (approximately 0.7 cm.^{2}, according to Gorlin[1]), the heart is unable to maintain an adequate output. Dizziness, syncope, and angina—with or without the specific changes of coronary arterial disease—become evident. The left ventricle under these circumstances must ultimately fail and, when this occurs, disaster follows in its wake. Pulmonary congestion and edema, right heart failure, and the ever-present threat of sudden death are the consequences of this uncompensated phase of aortic stenosis. Once this stage of diminished cardiac output with failure of the left ventricular myocardium is reached, the prognosis becomes grave and, as a rule, patients in this predicament have but a matter of months to live.

CLINICAL CLASSIFICATION OF AORTIC STENOSIS

The necessity for translating this inexorable chain of pathophysiologic events into a reasonable, practical, clinical classification, as a basis for surgical intervention is obvious. Such a clinical chain of events through which the average patient with aortic stenosis will pass is readily recognizable, although perhaps not clearly defined in the mind of the physician in the light of surgical advances. For a number of years we have used a system of staging (Table I) so that when the afflicted patient presents himself for evaluation, some estimation of the patient's status in relation to the timing of possible surgical intervention can be made.[2] In this way it is almost immediately possible, within reason, to recognize just where the patient may be in the over-all course of his disease syndrome. Thus, the patient can be more intelligently managed.

TABLE I. CLINICAL CLASSIFICATION OF AORTIC STENOSIS

STAGE	SYMPTOMS
	Before Left Ventricular Failure
I	Asymptomatic—murmur only
II	Fatigue, heart consciousness, palpitations
III	Syncope and/or angina—reduced cardiac output
	After Left Ventricular Failure
IV	Episodic pulmonary congestion
V	Frank congestive failure

Stage I is that period during which the typical systolic murmur of aortic stenosis is present, but before subjective symptoms have resulted. Stage II marks the onset of an ill-defined period in which the patient becomes subjectively aware of his forceful heart action, palpitations develop, easy fatigue becomes noticeable, and his attention may be called to the unusual visible pulsations in

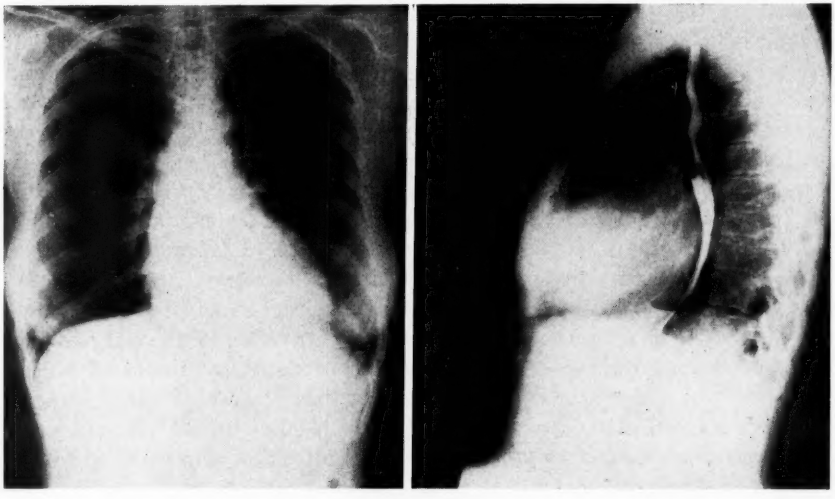


Fig. 2.—Typical cardiac configuration in aortic stenosis before onset of left ventricular failure. Note enlarged left ventricle and ascending aorta.

his supraclavicular and suprasternal notches. Admittedly, this stage is not specially characteristic of aortic stenosis per se, but when seen in the patient whose cardiac disease is that of aortic stenosis, the combination of the valvular changes and symptoms takes on added significance. It is an indication that the myocardium is beginning to feel the effects of an unyielding obstruction at the aortic level (Fig. 2). Stage III ushers in the obvious period of progressive disability due to a diminishingly effective cardiac output, evidenced cerebrally by spells of dizziness and actual syncope or myocardially by substernal discomfort, tightness, effort-angina indicative of an inadequate coronary flow. With beginning left ventricular failure, Stage IV is characterized by episodes of pulmonary congestion and edema, at first rather easily controlled by medical measures, but shortly to become refractory to even the most vigorous management. Stage V

connotes impending disaster and early demise for, in addition to left ventricular failure, superimposed right heart failure with hepatomegaly, ascites, and peripheral edema now labels the heart picture as one of almost irretrievable damage not to be reversed by any regime, medical or surgical.

With such a progression of easily recognizable clinical findings, it becomes obvious that if surgery is to be of any real benefit it must be performed at a time when the integrity of the left ventricle is still intact. Ideally, therefore, the patient in Stage II would represent the best candidate, for the mortality will be low and the functional result in terms of myocardial efficiency high. Certain it is that the patient should not be allowed to go beyond Stage III for as a rule, even at this relatively late hour, the myocardium has not failed. Once compensation of the left ventricle has been broken, the mortality resulting from surgery, whatever the technique, cannot fail to rise and the ultimate clinical result be reduced. Improved surgical techniques in very recent years have resulted in considerable salvage of patients in Stage IV and even in Stage V, but resort to surgery at this late date is like closing the barn door after the horse has been stolen. These facts will become more obvious with careful study of the clinical data from our own personal experience to be presented below.

HISTORY OF AORTIC VALVE SURGERY

The saga of aortic valve surgery has been a sporadic one. One of the earliest reported approaches to this valve was by Becker, an ophthalmologist (1872).[3] During his study of the retinal vascular changes seen in aortic insufficiency, he lacerated the aortic leaflets in animals, employing a glass rod passed down through the carotid artery into the ascending aorta. Tuffier,[4] in 1913, is credited with the first direct attack on aortic stenosis in a clinical patient. Although he proposed to incise the stenotic valve in a 36-year-old subject, at the time of surgery he was satisfied to invaginate the aortic wall into the valve orifice manually. The patient survived this procedure. In the light of present knowledge, it is hard to conceive that this method could have effectively relieved much of the stenosis encountered. Brock,[5] in 1947, visualized the aortic valve through the right subclavian artery using a specially designed cardioscope. His intent was to relieve the aortic stenosis by direct instrumentation but, after seeing the irregularly calcified valve which presented itself, it seemed obvious that direct valvotomy could not be carried out without undue risk. A major impetus to aortic valve surgery was provided by Smithy[6] who himself suffered from the ravages of the disease he hoped to be able to control. He concluded from his experimental work that the transventricular route to the aortic valve was preferable. Interestingly enough, he also chose this same approach to the mitral valve.

Experimental studies reported by Bailey, Glover, O'Neill, and Redondo Ramirez,[7] in 1950, included attempts to replace the valve by grafts, bypass procedures to circumvent the aortic valve, backward cutting punches to open the valve commissures, and various modifications of direct valvular incision (Fig. 3)

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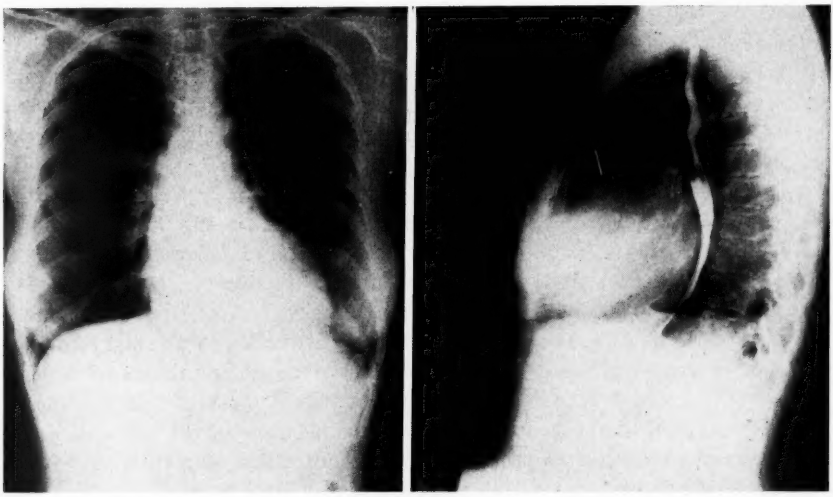

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as suggested by the work of Brock with regard to pulmonary valvotomy. Direct vision replacement of stenotic valves using a baffle clamp was reported by Litwak, Gadboys and their associates in 1952.[8] In 1953, Daniel and Scott[9] attempted to open stenotic aortic valves in 3 patients by direct insertion of a finger through the myocardium of the outflow tract of the left ventricle, but none of these patients survived.

Sarnoff and Case,[10] experimentally, used plastic valves of the Hufnagel type interposed between the left ventricular apex and the aorta to bypass the aortic valve but the high incidence of erythrocyte destruction and resultant emboli precluded clinical trial. Plastic and tissue diverticula sutured to the

Fig. 3.—An early approach to aortic stenosis using the right carotid artery for the introduction of a valve splitting instrument.

aortic wall just above the valve area have been used as tunnels to permit digital palpation and manipulation of the valve (Fig. 4). Glenn,[11] Bailey,[12] Swann,[13] and Harken[14] have expressed enthusiasm for this approach. Open cardiac surgical procedures employing hypothermia or extracorporeal bypass have received considerable attention in recent years. Valuable contributions to this aspect of the problem have been made by Swan,[15] Lewis, [16] and Lillehei[17] and their associates. Whereas this latter approach may ultimately be of considerable value in the congenital forms of aortic stenosis when employed at an early age, it would

seem to have little place in the surgery of acquired aortic stenosis until such a time as it becomes feasible to resect (totally) the damaged valve and to replace it with a plastic prosthesis.

TRANSVENTRICULAR APPROACH TO AORTIC STENOSIS

Despite the many ingenious approaches outlined, we have been convinced from the inception of modern valvular surgery that until hopelessly diseased and distorted aortic valves can be removed and replaced with impunity, the best functional result with the lowest risk to the patient will be obtained using a properly performed transventricular route. To support this contention, a consecutive series of 78 cases is presented, representing the two phases of transventricular commissurotomy techniques performed in this clinic. This series was

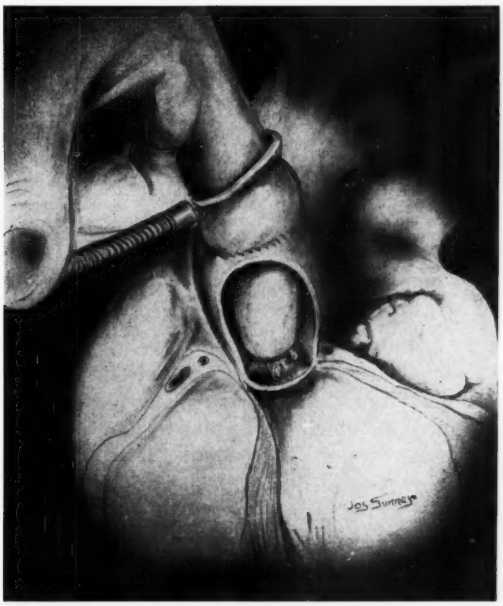

Fig. 4.—Approach to the aortic valve through the aorta itself employing a plastic sleeve sutured to the aortic wall for the insertion of exploring finger and instrument.

begun in March, 1951, and for purposes of this presentation includes every patient operated upon through June of 1957. The age range in this group was from 4 to 57. In 24 of these patients, combined mitral and arotic commissurotomies were performed. In one instance, an associated coarctation of the aorta was resected with end-to-end anastomosis, after which the aortic commissurotomy was accomplished. The great majority of these operations were carried out in patients in the far-advanced stages of their disease as seen in Table II. Seventy of the 78 patients had moderate to marked calcification of the aortic valve. Thirteen were in permanent atrial fibrillation (ten of these were suffering from both mitral and aortic stenosis).

TABLE II. ANALYSIS OF LATE DEATHS FOLLOWING AORTIC COMMISSUROTOMY

STAGE	NUMBER OF CASES	SURVIVAL PERIOD (MO.)	CAUSE OF DEATH
Old Technique			
II	1	10	Subdural hematoma
III	4	5	Congestive heart failure
		24	Congestive heart failure
		17	Congestive heart failure
		35	Coronary occlusion
IV	1	8	Sudden collapse
V	2		
		41	Congestive heart failure
		5	Pulmonary embolus
New Technique			
III	2	12	Congestive heart failure
		7	Congestive heart failure with aortic insufficiency
IV	3	9	Suicide
		14	Congestive heart failure
		1	Congestive heart failure with aortic insufficiency
V	2	4	Active rheumatic heart disease with bacteremia
		2	Cardiac arrest
	15	1 to 41	

The first 37 cases in this series were operated upon by the old original technique as reported in 1952,[18] employing a large three-bladed expandable split-dilator (Larzelere-Donaldson design) inserted through a widely purse-stringed avascular area near the apex of the left ventricle (Fig. 5). This method has long since been abandoned because of the unwieldiness of the instrument, the unnecessary myocardial trauma, and the excessive blood loss which it occasioned. The remaining 41 patients, operated upon since June of 1955, were operated upon by a greatly refined technique using a dilator demonstrated by Sir Russell Brock.[5] This instrument is light but exceptionally strong, is no larger in caliber than the usual lead pencil, and its expandable head is equipped with two parallel blades rather than three as were formerly used (Fig. 6). It does not cut but dilates the valvular orifice to cause splitting of one or two commissures, occasionally three. Briefly, the technique as employed is as follows: a left posterolateral approach is used entering the pleura through the widely opened fifth interspace giving excellent exposure to the left ventricle and indeed the entire left heart. The pericardium is opened vertically, anterior and parallel to the phrenic nerve from the level of the aortic arch to the diaphragm, thus widely exposing the left heart and great vessels. The aortic ring and its contained valvular tissues are explored externally by palpation and, in most instances, considerable information regarding the type of valve to be encountered, including the degree of calcification and its involvement of the surounding aortic wall or myocardium, can be gained. The ascending aorta is likewise palpated for an estimation of the degree of thrill present and the extent of the post-stenotic dilatation of that structure. Direct pressures are taken from within the ascending aorta, the left ventricle and the left atrium. The systolic aortic

Fig. 5.—The original large sized three-bladed aortic valve dilator for transventricular approach to the valve. Note guide wire in the aorta over which the instrument is threaded and the wide myocardial purse-string suture employed for hemostasis during manipulation.

Fig. 6.—The small but powerful two-bladed dilator presently used for the transventricular approach.

gradient indicative of a block has ranged from 30 to 250 mm. Hg in this series. There are those who feel that the measurement of these pressures under anesthesia and in the open chest in this manner is of little value and is not an accurate hemodynamic measurement. While this may be true in the strict academic sense, for practical purposes these measurements are of the greatest value for not only do they give one an estimation of the degree of block, but by comparing pre- and postcommissurotomy pressures very definite knowledge of the efficacy of the procedure performed can be obtained. Indeed most of the pressure tracings taken in this manner agree amazingly closely to those taken by left heart catheterization prior to surgery, so that we believe that this type of hemodynamic control is absolutely essential to the proper performance of aortic valve surgery. A Statham P23D transducer, using an atmospheric O point, is the type of gauge employed. The head vessels are not isolated or constricted in any manner during the operative procedure. In an experience with over one thousand mitral commissurotomies, no diminution in the incidence of cerebral emboli could be recognized between patients in our series who did not have temporary occlusion of these arteries and in the series of others in which this measure was employed. Indeed, we feel that such a measure is more dangerous than no occlusion at all. Strangely, although one would expect a much higher incidence of emboli in aortic stenosis in which the valves are almost routinely heavily calcified, embolism, in our experience, following aortic commissurotomy has been rare, considerably less than that seen in mitral commissurotomy.

A 1 cm. incision is made halfway through the thickened left ventricular myocardium, so as not to enter the lumen, in an avascular area near the apex of the left ventricle. No bleeding results from this initial incision. No purse-string suture and no stay sutures of any type are used for they tend to damage or crush the myocardium or often pull out with resultant laceration of this muscle. With this technique, bleeding is so minimal that such measures are not needed. It is vital to recognize this fact for this is one of the major refinements responsible for present improved mortality statistics. At this point all is in readiness for the passage of the aortic dilator. The second major feature in this improved technique occurs at this point. All operative manipulation ceases, and for 5 minutes by the clock the anesthetist completely expands the lungs and oxygenates the patient to the fullest. It is felt that this lowers the possibility of myocardial ischemia and resultant arrhythmias during the period of manipulation and, since observance of this feature, arrhythmias other than an occasional premature contraction have rarely been seen. Exposure is then obtained and the two-bladed Brock dilator is gently pressed into the previously initiated incision. As the myocardium contracts and relaxes the dilator held against it with slight pressure dissects its own way through the myocardium into the lumen of the left ventricle. The dilator is then guided upward and passed through the stenotic orifice, being aided in this maneuver by the external hand which is placed about the aortic ring as a target at which to aim. The entire passage of the instrument is done slowly and with deliberation and in a very gentle manner so that there is no jabbing or thrusting under pressure. It is very important to realize that the initial passage toward the aortic valve is

downward toward the floor, rather than immediately upward toward the head. This point might be forgotten in the excitement of the operative maneuver. To direct it immediately upward is to impinge upon the interventricular septum or myocardium leading to injury or arrhythmia. A few seconds may be necessary to find the exact orifice for often one impinges upon exuberant calcified plaques and induration occurring in the outflow tract just proximal to the valve or protruding down from the valve itself like thickened stalactites. With care and deliberation there has been no instance where the valve orifice could not easily be entered. The hand (left) which is palpating the aortic ring externally can appreciate the presence of the dilator within the orifice, and can feel half of the dilating head extending out into the aorta itself. When the dilator is properly placed, it is slowly opened to its full extent, first in one plane, and then is carefully rotated in two other planes to ensure the maximum separation of fused valve elements at the commissures. No attempt is made to recognize anatomically each commissure, for these valves are so badly diseased and distorted that such recognition is impossible. The dilator is merely opened where it will, in the three planes mentioned, and routinely the fused valve cone will split at its commissures. One and usually two commissures are readily opened in this manner, occasionally three, although this latter is unusual. One can feel the valve split and crunch as its calcific fusion is split asunder. It is the source of utter amazement that all of these patients do not suffer clinical emboli at this time for, without question, innumerable sandlike particles are dispersed into the circulation but, as stated above, evidence of clinical emboli during the postoperative period rarely is seen. Should the head of the dilator be felt to slip upward into the aorta or backward into the ventricle because of the toughness of the tissues, thereby modifying the splitting compression, the maneuver is repeated rapidly at two or three levels along the outflow tract or up in the aorta to ensure maximum separation of valvular elements. The instrument is closed and slowly withdrawn. Pressure over the small, linear, 1 cm. incision with the finger and a sponge for 1 or 2 minutes will in almost every instance cause complete cessation of bleeding, and with careful manipulation the tiny incision has not been enlarged upward or downward, so that an absolute minimum of myocardium will have been injured. When the heart rate and action is stable and the blood pressure has returned to the same level as it was prior to commissurotomy, the pressures are repeated in the aorta, left ventricle, and left atrium. In most instances, the gradient will have dropped dramatically. In others it will not seem to be entirely effective so that the dilator may be replaced two, three, and even four times, for it is well tolerated as a rule. Occasionally the head of the dilator is removed and another of the same design but larger is attached, a modification of Brock's instrument. This has been effective in further reducing the gradient. In many instances, the gradient has been all but abolished and in many others has dropped to less than 30 mm. Hg. One must remember that often it is obviously impossible to abolish completely the block at the aortic valve level because of the physical presence of large amounts of calcium, the presence of which at this location alone precludes such an accomplishment. Such a valve cannot retract or open completely. When the hopeless nature of

the disease present is fully appreciated it will be recognized that the surgeon is attempting merely to improve flow to a maximum degree and not to restore the valve or its action to a normal state, obviously impossible. Most valves in acquired stenosis have lost all semblance of a valve for there is little if any pliability to assure valve action. One is therefore attempting to provide an increased orifice and hopes that some measure of valve motion may be restored, depending upon the individual nature of the particular patient's pathologic changes. Not more than this can be expected until the day when this entire calcified structure can be resected and replaced by a suitable prosthesis.

The tiny myocardial wound is reinforced with two or three heavy sutures, the pericardium is closed completely, a dependent incision having been placed posteriorly for drainage into the pleural space. The chest is closed in the usual manner, using catheter drainage of the pleural space routinely. Recently, in those patients whose myocardium has deteriorated to an exceptional degree or in whom angina pectoris was a prominent preoperative symptom, the internal mammary artery is divided from within the left chest at the level of the second anterior interspace and, after the chest is closed and the patient placed on his back, the right internal mammary artery is divided through a small secondary incision. This feature has been added because of the marked symptomatic improvement obtained in many patients suffering from angina pectoris secondary to coronary insufficiency when this procedure has been employed.[19]

HEMODYNAMIC OBSERVATIONS

In this series of cases, cardiac and arterial catheterizations were carried out preoperatively in 47 instances, this form of study being used only sporadically before June, 1954. When possible, and this was accomplished not infrequently, the arterial catheter placed into the arch of the aorta was passed retrogradely across the aortic valve into the left ventricle. Aortic and brachial artery pressure tracing routinely showed contours suggestive of aortic stenosis with a typical curve, indicating a delayed and notched ejection phase. Cardiac outputs (Fick) varied from 2.10 to 5.96 L. per minute, averaging 3.77 L. per minute for the catheterized series.

As stated, since December, 1954, direct pressure studies have been routinely made at the time of surgery (Fig. 7). The differential pressures before aortic commissurotomy have agreed systematically with the preoperative valvular gradients obtained. The precommissurotomy systolic gradients across the aortic valve have varied from 8 mm. Hg to 250 mm. Hg, averaging 78 mm. Hg. (The patient having only an 8 mm. Hg gradient was operated upon mistakenly, for although her studies and symptoms were characteristic of aortic stenosis, at operation she proved to be suffering from idiopathic hypertrophy of the left ventricle without valvular component.) Postcommissurotomy aortic valve gradients range from 0 to 75 mm. Hg, averaging 27 mm. Hg.

A slightly greater hemodynamic change occurred in the patients who as yet had not gone into failure prior to surgery. Of 19 patients in Stage II or III having operative pressure studies, the left ventricle-aortic systolic gradient

averaged 83 mm. Hg before and 29 mm. Hg after commissurotomy, a reduction of 54 mm. Hg, obviously of marked significance. The average systolic gradient in 24 patients in Stages IV and V (after left ventricular failure) dropped from 70 mm. Hg to 28 mm. Hg following commissurotomy (a 42 mm. Hg reduction).

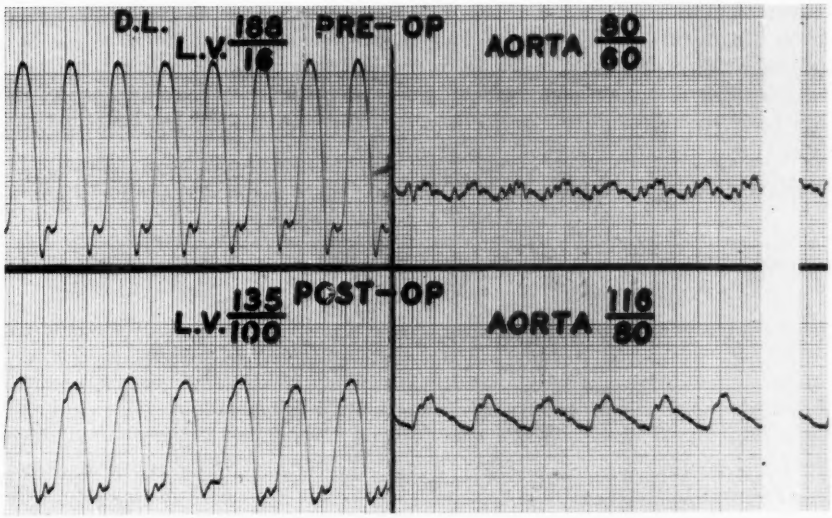

Fig. 7.—Pre- and postaortic commissurotomy pressure tracings taken within the left ventricle and aorta. Note the highly satisfactory reduction of the aortic valve block (from 108 mm. Hg to 19 mm. Hg).

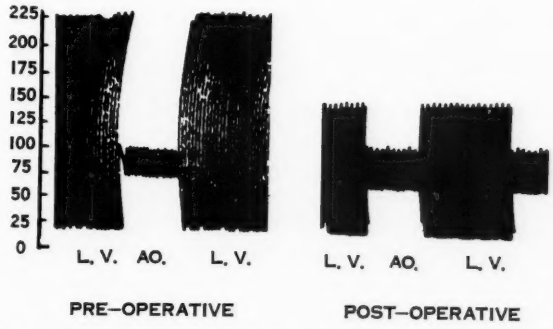

Fig. 8.—Typical hemodynamic response within left ventricle and aorta to transventricular aortic commissurotomy by the method described (recorded at 1 mm./sec. paper speed).

It is obvious that complete abolition of the valvular gradient rarely ensues for reasons considered above, namely, the nature of the pathologic changes present (Fig. 8). These hemodynamic results are an improvement over those which have been published (very few reported) by all other aortic valvular approaches (Fig. 9).

MORTALITY

As yet no appreciable series of patients in whom extracorporeal methods were employed in the relief of acquired aortic stenosis have appeared in the literature, but in a few small series reported the mortality rate has ranged from 50 to 100 per cent. This obviously represents a developmental phase which should be short lived once adequate valvular replacement becomes possible. In the recent past, Bailey has reported an operative mortality of 15.1 per cent using the aortic approach to the valve, but few have attained these results. On the other hand, Brock's success with the transventricular route has been very impressive. His over-all operative mortality has been 16 per cent but in his most recent 63 reported cases, there were but 4 deaths, an extremely respectable 6 per cent mortality.[5]

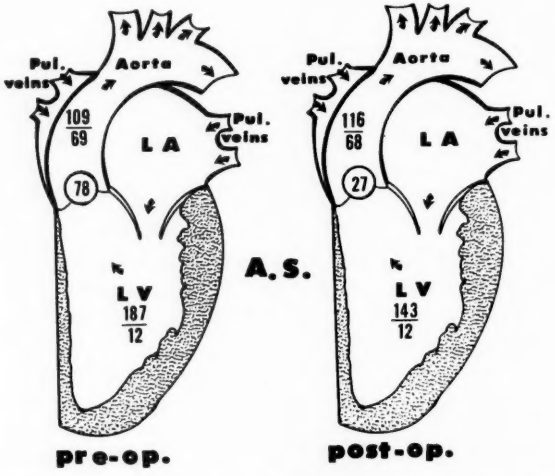

Fig. 9.—Average preoperative and postoperative gradients for the entire series of patients with aortic stenosis.

The deaths in this series of 78 patients are divided into two categories.

Operative Deaths.—Those patients who died in the hospital during or after surgery up to 1 month.

Of the 37 patients operated upon by the older obsolete technique, there were 17 deaths, a mortality rate of 46 per cent, obviously intolerable.

In the more recent group of 41 patients operated upon by the present technique outlined above (since June, 1955), there have been but 2 deaths or a very respectable mortality rate of 4.9 per cent.

In both groups, 17 of the 19 operative fatalities were in patients in the late stages of their disease, having been in repeated cardiac failure (Stage IV or V), and 13 were in Stage V. There were 13 deaths of cardiac origin (mechanism deaths); the remainder occurred during the postoperative hospitalization from congestive heart failure (3), myocardial infarction (1), operative embolus (1), and cerebral vascular accident occurring 10 days postoperatively (1).

Late Deaths.—In the entire series, 15 patients have died since leaving the hospital (1 to 41 months after surgery). Eight of these late deaths occurred in patients who had been subjected to the old operative technique, 7 deaths were in the group who underwent the new operative technique. These data are presented in Table II. It should be added that one of the patients who died at a late date after surgery did not die from his cardiac disease per se for he committed suicide. This was strange for he had obtained a good functional result but lived in fear and dread of his disease potential and could no longer face reality because of this fear.

Although many of these patients have died in various parts of the country, autopsies have been obtained in 16. Coronary occlusion was demonstrated in 2 of these. The coronary arteries were patent in the remaining although some evidence of atherosclerosis was as a rule observed.

CLINICAL RESULTS

The present status of patients presently living has been ascertained by either direct examination or from specific reports from their referring physicians. Twelve of the 37 patients operated upon by the older technique are living to this day (3 to 7 years) and all are distinctly improved. Their ability to work, walk, and indulge in mild exercise is distinctly better than it had been in any time during the years immediately before surgery, so that for them the operation was well worth while. One patient has occasional dyspnea on exertion and another takes infrequent nitroglycerin tablets for chest pain but otherwise remains improved.

Thirty-three of the 41 patients operated upon by the new technique June 1, 1955, to June 1, 1957) have been analyzed, there being a reasonable length of observation of a year or more for analysis. Twenty-five of these are alive and 22 are very definitely improved. These have shown definite symptomatic improvement and in them again surgery has been of great benefit. To be sure, these patients realize that they do not have new hearts or new valves, but are now living with their diseased condition in a vastly improved state but require careful medical observation and management. None of this group has shown signs or symptoms of aortic insufficiency. Nineteen of them have returned to various degrees of work, ranging from housework to welding. The incidence of syncope, dizziness, shortness of breath, chest pain, orthopnea, and recurrent congestive failure has been uniformly improved or in most instances abolished. Thus, 22 of this group of 33 patients in this newer group have been very materially helped by surgery (67 per cent). The remaining 8 patients of the new group of 41 are all living and improved within the first year but not enough time has elapsed to include them in the larger group of late results.

Three patients in the newer group have not been benefited, although they are still alive. One is the patient operated upon with an erroneous diagnosis which proved at operation to be that of idiopathic hypertrophy of the left ventricle without valvular involvement. Another patient, although able to

work intermittently, is orthopneic, has angina, and dizziness, and is unable to climb stairs. The third patient, while improved from the cardiac standpoint, is still recuperating from an operative cerebral embolus (Table III).

TABLE III. LONG-TERM RESULTS FOLLOWING AORTIC COMMISSUROTOMY

STAGE	NUMBER OF CASES	SURVIVAL PERIOD (MO.)	PRESENT STATUS
Old Technique			
II	2	32 and 57	Improved
III	4	38 to 63	2 Improved 2 Excellent
IV	4	37 to 64	Improved
V	2	47 and 65	Improved
Total	12		
New Technique			
II	1	16	Excellent
III	13	8 to 31	5 Excellent 7 Improved 1 Unimproved
		9 to 22	3 Excellent 5 Improved
IV	9		1 Unimproved
V	2	10 to 27	Improved
Total	25		

DISCUSSION

It is thought that the technique employing the transventricular route is the best possible form of surgical palliation for aortic stenosis at the present time and until, by open-heart methods, the valve can be replaced. When one considers the pathologic changes with which the surgeon must cope, and realize that the pathophysiologic changes resulting from the disease are inexorable and follow a rather set pattern, it becomes obvious that the only way to improve these results is by earlier selection of patients for surgery. Because, in recent years, most physicians have had considerably more experience with the problems of mitral stenosis than they have had with those of aortic stenosis, much confusion exists as to what constitutes the proper indications for surgery in aortic stenosis. Patients are being referred for operation in aortic stenosis on the basis of indications employed in mitral stenosis. This practice must terminate for the two conditions present major differences. The difference lies in the effects which these two valvular conditions exert upon the integrity of the left ventricular myocardium. The left ventricle is the *cornerstone of the heart.* Despite numerous and repeated bouts of pulmonary edema and right ventricular failure, the left ventricle in mitral stenosis has been spared and remains as a strong and effective chamber. Actually, valvular obstruction at the mitral level has provided a measure of protection. It follows then that decompensation in mitral stenosis is essentially right heart failure and not left. With the establishment of more normal mitral valvular mechanics by surgery, the left

ventricle can resume its fundamental role of maintaining total circulation effectively. Thus, if one waits in mitral stenosis for numerous bouts of congestive failure to occur, these patients may still be improved to a high degree mainly because of the highly effective action of the left ventricular myocardium. These facts have influenced the physician's thinking and have determined his referral of all valvular patients for operation. The delay in waiting for the signs and symptoms of decompensation to appear is the greatest single factor in the poorer results obtained in the surgery of aortic stenosis.

It must be realized and repeatedly emphasized that from the very beginnings of aortic stenosis the brunt of the obstructive effect is absorbed by the left ventricle itself. It follows, therefore, that when the patient with aortic stenosis shows signs of decompensation, his cardiac failure means left ventricular failure. One cannot wait in aortic stenosis, therefore, until pulmonary congestion or frank congestive failure have developed, for myocardial reserve will then be all but gone. This distinct difference between the effect of mitral stenosis and aortic stenosis must be appreciated to the fullest extent before patients suffering from the latter condition will receive their best possible chance for survival and improvement. One has only to appreciate the fact that 17 of the 19 operative deaths in this series were in patients who had already experienced left ventricular failure to comprehend that new and realistic criteria for operation must be established if these patients are to be given a fighting chance. Furthermore, once surgery has been performed, these patients must not be sent out into the world to fend for themselves, medically speaking. They need careful medical advice and management indefinitely for the future. Even in those patients who have obtained the best hemodynamic response at the time of surgery, one must appreciate the fact that the left ventricle for years has been taxed to its uttermost and now needs intelligent careful support for its remaining lifetime. To recapitulate: when the nature of the pathologic changes in aortic stenosis is fully appreciated, when the physiologic and myocardial effects of these changes are recognized, when the patient is referred for operation prior to left ventricular failure, and when surgery is performed by a swift and simple technique, then, and only then, will patients suffering from this most difficult of all valvular lesions be given their best chance for survival and maximal functional improvement.

SUMMARY

1. Seventy-eight patients with aortic stenosis have been operated upon by the transventricular approach since March, 1951.

2. The first 37 patients were operated upon using a now outmoded technique, with 46 per cent operative mortality.

3. The last 41 patients have had split-dilatation of the valve by the present method, with 4.9 per cent operative mortality.

4. The average systolic peak gradient across the valve has been reduced from 78 mm. Hg to 27 mm. Hg.

5. There have been 15 late deaths, 8 in the earlier group, and 7 in the group operated upon by the present operative technique.

6. Thirty-four of 37 surviving patients operated on more than 1 year ago have shown definite improvement from their surgery.

7. It is believed that, until the diseased aortic valve can be totally removed and replaced, the transventricular approach offers the best form of surgical palliation with the least possible risk.

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CLOTTING DEVIATIONS IN MAN ASSOCIATED WITH OPEN-HEART SURGERY DURING HYPOTHERMIA

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INTRODUCTION

OPEN-HEART surgery performed under hypothermic anesthesia or with the utilization of an extracorporeal circulation subjects the individual to a stress situation in which the blood is subjected to physical changes in its environment. It is conceivable that the manipulation of tissues, such as the lungs, during the operative procedure may effect the physiologic equilibrium of the clotting and fibrinolytic systems by the release of activators and/or inhibitors into the circulating blood. It is also possible that hypothermia or the use of the pump-oxygenator may induce or enhance changes in the mechanism of clotting. Consequently, we have investigated the nature of the clotting deviations which may occur under these conditions. This communication presents our investigation of the changes in the clotting mechanism during open-heart surgery under hypothermic anesthesia. The changes associated with the utilization of the extracorporeal pump-oxygenator are presented in another publication.[10]

PROCEDURE

Citrated plasma (one part sodium citricum tribasicum U.S.P., 3.8 per cent, to four parts of blood) was used throughout the study. The blood was obtained by a two-syringe technique and the plasma was immediately separated by centrifuging the specimen for 5 minutes at full speed in an International clinical centrifuge. The plasma was processed within 30 minutes after the specimen was obtained, and ordinary Pyrex glassware* was used during the determination. The average lowest temperature obtained during hypothermic anesthesia was 28° C. The following clotting tests were performed at both 28° C. and at the conventional test temperature of 37° C.

1. *Recalcification Time.*—This test indicates the over-all coagulability of the blood. In a test tube, 0.03 c.c. of 0.5M $CaCl_2$ was carefully blown into 0.3 c.c. of prewarmed plasma. The end point indicating completion of clotting (recalcification time) is reached when the tube can be tilted to an angle over

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*In most cases.

90 degrees without spilling liquid. Our modification of this test is more sensitive for deviations than the Lee-White time, and undesirable dilutions of plasma are kept at a minimum. This plasma dilution is brought about by the conventional recalcification procedures and disrupts the normal relationship of inhibitors and activators in the plasma. The normal recalcification times at 37° C. are 6 to 10 minutes.

2. *Prothrombin Consumption.*—The prothrombin consumption reflects abnormalities in the first phase of clotting; that is, the thromboplastin formation. This is measured by our own personal method herein described for the first time. The same specimen employed in the recalcification time study is used. Sixty minutes after completion of clotting, the clot was carefully but firmly wound around a small wooden applicator stick and removed from its serum. Then 0.06 c.c. of 3.8 per cent sodium citrate was added to the serum. After an additional 30 minutes (i.e., 90 minutes after the completion of clotting), the prothrombin not consumed during the clotting process was determined in the serum. To 0.1 c.c. of thromboplastin suspension (see below), 0.1 c.c. of fibrinogen solution was added. The fibrinogen solution was made up of 1 per cent bovine fibrinogen Armour dissolved in a mixture of one part of sodium barbital acetate buffer, pH 7.4, and four parts of sodium chloride (0.85 per cent) stirred frequently for 10 minutes with 10 mg. of barium sulfate per cubic centimeter, and the clear supernatant fluid frozen in aliquots. The mixture was prewarmed for 2 minutes at 37° C., at which temperature the final steps of the test were always done, and 0.1 c.c. of the test serum was then blown into it. The clotting time was determined with the sera remaining after clotting at 37° C. and at 28° C. The serum may be stored in the refrigerator for several hours before testing. The normal values for clotting at 37° C. are 40 to 60 seconds.

3. *Prothrombin Activity.*—This conventional one-stage prothrombin time is the result of the combined influence of the activity of prothrombin, factors V and VII (and inhibitors, if present). The gray matter of a fresh human brain, carefully cleaned of meninges and blood traces, is suspended in equal parts by weight of 0.85 per cent sodium chloride by means of a blender, and the mixture frozen in aliquots. An aliquot of this suspension, diluted with ten parts of 0.05M calcium chloride, is used as thromboplastin. The prothrombin activity was read in the usual manner from a plotting graph obtained from the thromboplastin batch which had preserved its full activity for months.

4. *Thrombin Time.*—This test essentially measures the activity of heparin and heparin-like inhibitors. One thousand units of bovine thrombin were dissolved in 5 c.c. of 50 per cent glycerol and stored at –10° C. One part of this solution was diluted in a siliconized test tube with 16 parts of buffered saline (see Prothrombin Consumption) with a pipette, 0.025 c.c. of this resulting solution was blown into 0.2 c.c. of prewarmed (37° C.) citrated plasma and the clotting times were measured. Care was taken to use the thrombin solution immediately after its preparation. Each batch of thrombin was tested on normal plasma. The normal values range from 11 to 13 seconds.

5. *Fibrinolysis.*—The following test for fibrinolytic tendency can demonstrate an increased tendency toward fibrinolysis which cannot be discovered in the process of watching "normal" clots. One part of citrated plasma was diluted with 18 parts of buffered saline and clotted with 0.01 c.c. of undiluted thrombin solution. The clot dissolution time was measured. Undiluted plasma, 0.3 c.c., was used and clotted with the same amount of thrombin. Normal values are present when there is no fibrinolysis in 24 hours either in diluted or in undiluted plasma upon incubation. This method is not accurate when used with heparinized blood. In its stead, in studies on heparinized patients, the euglobulin lysis method, which also gives information about the fibrinolytic activity of heparinized blood,[11] is to be preferred.

6. *Thrombelastography.*—This procedure permits continuous recording of fibrin formation and fibrinolysis and gives a good estimate of the firmness of the clot. Our technique has been described elsewhere.[8]

CLINICAL APPLICATION

There were 11 patients involved in this study. Eight of these patients underwent open-heart surgery in association with hypothermic anesthesia while the remaining 3 patients had their procedures performed under normothermia. In general, the anesthetic agents used were cyclopropane-ether combinations and, in some cases, pentobarbital was administered for induction. Curare and Prostigmin were given in most instances, and transfusions of stored ACD blood were administered at the beginning of the procedure with the rate of infusion adjusted to the needs of the patient.

The patients involved in this study are presented in Table I which includes the age and sex of the patient, the type of operative procedure, the duration

TABLE I

NO.	PATIENT	AGE	SEX	OPERATIVE PROCEDURE	OCCLUSION (MIN.)	LOW TEMPERATURE (DEGREE C.)	BLOOD (C.C.)
1	E. C.	8	M	Transventricular infundibulectomy; pulmonary valvulotomy	5.20	28.0	400
2	B. S.	34	M	Division of patent ductus arteriosus	—	27.0	1,500
3	M. M.	22	F	Suture closure of atrial septal defect	5.20	28.0	1,500
4	R. E.	14	M	Suture closure of atrial septal defect	7.10	28.0	1,500
5	W. S.	24	F	Suture closure of atrial septal defect	5.50	26.5	7,800
6	M. R.	35	F	Suture closure of atrial septal defect	6.15	27.4	2,500
7	C. Z.	3	M	Pulmonary valvuloplasty	3.00	29.0	300
8	G. M.	28	M	Suture closure of atrial septal defect	4.20	29.0	2,000
9	R. H.	6	M	Resection of coarctation of the aorta	—	—	450
10	M. F.	31	F	Mitral commissurotomy	—	—	800
11	W. J.	48	M	Thoracoplasty, 3rd stage	—	—	1,700

TABLE

PATIENT	RECTAL TEMPERATURE (DEGREES C.)					RECALCIFICATION TIME (MIN.)					SERUM PROTHROMBIN TIME (SEC.)				
	BA*	AA†	AC‡	DO§	CC‖	BA	AA	AC	DO	CC	BA	AA	AC	DO	CC
1	37.2		28.0	28.0	32.0										
37°						5.00		5.30	4.30	5.30	62	—	72	59	116
28°								7.00	4.20	5.45	—	—	62	74	78
2		36.5	32.8	28.0	33.0										
37°							8.30	29.00	19.00	9.30	—	43	35	47	49
28°							7.20	14.00	14.00	9.45	—	47	44	55	60
3	37.0	36.8	30.0	28.9	27.5										
37°						8.00	11.00	8.30	7.00	7.00	42	43	57	58	62
28°						8.30	10.30	7.30	9.45	9.00	54	57	49	55	68
4	37.0	37.5	31.5	28.0	30.0										
37°						5.30	4.00	6.15	4.45	9.00	73	63	62	124	80
28°								5.10	5.30	5.00	—	—	115	114	119
5	37.0	37.0	30.0	27.5	33.0										
37°						8.00	5.00	5.00	5.00	6.00	53	54	53	57	84
28°						6.30	6.30	7.15	7.00	5.30	130	75	79	80	115
6	37.0	37.2	30.0	27.5	32.5										
37°						7.30	8.00	7.30	8.30	6.00	62	65	68	74	79
28°						7.00	8.00	6.30	8.00	7.00	73	76	72	85	94
7			32.2	27.3	36.8										
37°						14.00	19.00	18.30	12.00	10.00	28	33	40	76	75
28°						10.30	13.30	15.00	10.30	8.00	45	64	72	69	99
8	37.2	37.0	32.0	29.5	31.0										
37°						8.00	7.00	4.15	5.30	6.00	57	44	50	65	53
28°						12.00	7.00	4.00	7.00	6.00	109	88	96	88	81
9															
37°						6.00	4.15	—	5.00	6.00	60	69	—	—	140
28°						—	—	—	—	—	—	—	—	—	—
10															
37°						31.00	32.00	—	18.00	16.30	31	32	—	28	35
28°						16.00	17.30	—	12.00	13.30	62	54	—	68	65
11															
37°						14.00	17.00	—	15.00	20.00	44	27	—	40	41
28°						9.00	13.00	—	18.00	12.00	64	60	—	76	80

*BA=Before anesthesia; †AA=After anesthesia; ‡AC=After cooling; §DO=During operation;

of inflow-outflow occlusion, the lowest temperature obtained during hypothermia, and the amount of blood infused.

The tests of the clotting mechanism were done at both 37° C. and 28° C. Attempts were made to obtain the blood specimens before anesthesia (BA), after anesthesia but before cooling (AA), after cooling but before operation (BO), during operation immediately after occlusion (DO), and after closure of the chest (CC) in all hypothermic patients (1 through 8); and before anesthesia (BA), after anesthesia (AA), during operation (DO), and after closure of the chest in normothermic patients (9 through 11). In some instances, additional clotting tests were performed on the following day. Our findings for these various determinations are presented in Table II along with the corresponding patient number and the lowest temperature recorded during the period of hypothermia.

PROTHROMBIN ACTIVITY (PER CENT)					THROMBIN TIME (SEC.)					FIBRINOLYSIS (DILUTED PLASMA)					LOWEST TEMPERATURE (DEGREES C.)
BA	AA	AC	DO	CC	BA	AA	AC	DO	CC	BA	AA	AC	DO	CC	
60	—	53	61	45	—	—	—	—	—	—	—	+++	—	—	28.0
—	—	—	—	—	—	—	—	—	—						
—	50	45	43	40	—	—	—	—	—	—	++	+++	+++	+++	28.0
—	—	—	—	—	—	—	—	—	—	—	—	—	—	+++	
48	44	43	40	37	14.8	16.2	19.5	17.6	18.9	—	—	—	+++	—	28.0
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
50	60	55	40	40	14.7	13.5	16.2	16.5	14.2	—	—	—	+++	—	28.0
—	—	—	—	—	—	—	—	—	—	—	—	—	+++	—	
65	60	60	53	40	14.6	15.5	19.5	17.2	16.5	—	—	—	—	+++	26.5
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
52	51	41	35	33	11.4	14.8	15.2	19.7	14.0	—	—	—	—	—	27.4
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
55	44	43	41	39	12.3	15.0	14.3	14.0	13.4	—	—	—	+++	—	29.0
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
65	55	45	42	43	10.6	12.5	9.8	11.4	12.0	—	—	—	—	—	29.0
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
55	57	—	82	82	12.0	13.0	—	13.3	10.2	—	—	—	—	—	
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
45	44	—	43	53	12.0	14.6	—	15.0	11.1	—	—	—	—	++	
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
45	55	—	68	63	17.9	18.0	—	17.0	17.7	—	+++	—	—	+++	
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	

CC=After closure of chest.

RESULTS

During the course of open-heart surgery under hypothermic anesthesia, the *recalcification time* exhibited a trend toward shortening rather than toward prolongation. This applied equally to the tests performed not only at 37° C. but also to those at 28° C. In only 1 of 8 patients could a clear-cut but transient prolongation of the recalcification time be observed during the first phase of cooling (patient 2). It is interesting to note that this prolongation was more pronounced in the test done at 37° C. than at 28° C. Consequently, it is doubtful whether the clotting mechanism of this particular patient was even affected since the body temperature was 33° C. at the time of the determination. The lowest temperature reached was 27° C., but the recalcification time gradually returned to normal value. In general, the recalcification times done at 28° C.

were shorter than the ones of the same plasma specimen done at 37° C. This is well demonstrated in patients 7, 10, and 11 who had long recalcification times at the beginning of the procedure.

The *prothrombin consumption* showed a pattern which corresponds to that of the recalcification time; that is, the prothrombin consumption, reflected by the longer prothrombin serum times, was more complete in the specimens incubated at 28° C. than in those incubated at 37° C. This applied to 40 out of 42 prothrombin consumption tests done at both temperatures. In 2 patients (7 and 10), who had a poor prothrombin consumption at 37° C., the consumption was normalized when the test was performed at 28° C. Generally, cooling failed to bring about any reduction of the prothrombin consumption below the normal values for 37° C. When the blood sample was incubated at 37° C., a decrease in prothrombin consumption in the sample taken after cooling was observed in only 1 patient (patient 2). This was the same patient in whom cooling induced a transient prolongation of the recalcification time for the same specimen. Test tube studies with plasma from normal persons proved that the prolonged serum prothrombin times of samples incubated at 28° C., as compared with those incubated at 37° C., is due to a real and not an apparent increase in thromboplastin formation. It can be said that the prothrombin consumption measured at both temperatures did not show any striking changes in either hypothermic or normothermic patients, except for a trend toward exaggerated prothrombin consumption near the completion of the operation.

The *prothrombin activity* showed a persistent drop in all 8 of the hypothermic patients during the operation. However, there was a rise in the activity on the day following the procedure. This decrease was not observed in the normothermic group. In fact, in all 3 of these patients, a trend toward an increase of the prothrombin activity was noted during the operation and this was even more pronounced on the day after surgery.

The *thrombin time* frequently exhibited a tendency toward prolongation. In patients, 3, 5 and 6, the thrombin time was shifted during the operation to values which we consider as abnormal. The maximum rise was seen after cooling in patients 3 and 5, and after occlusion in patient 6. Patient 3 had a transient episode of auricular fibrillation during the procedure, but showed no evidence of hemorrhagic manifestation. Patient 6 died the day after the operation after two episodes of ventricular fibrillation. At autopsy, there were large, scattered hemorrhagic areas in the lungs and recent thromboses of both small and large vessels. The rise in thrombin time was accompanied by a very definite bleeding tendency in patient 5. The resulting trends of the clotting tests on this patient are reproduced graphically in Fig. 1.

Coagulograms obtained on this patient clearly indicated that there was a delay in the measurable onset of fibrin formation in the sample obtained after cooling (AC). This same period exhibited the most abnormal thrombin time values. After the thrombin times returned to normal, there was no further evidence of such a delay. Fig. 2 demonstrates this fact clearly.

The tendency toward increased *fibrinolysis* was observed in 8 patients. In 3 of the hypothermic group, this fibrinolytic tendency was increased only after occlusion. The most persistent lysis tendency was noted in patient 2, in whom many adhesions around the vascular truncus had to be separated. In both patients 2 and 4, the increased lysis tendency was also observed in specimens incubated at 28° C. In none of the patients could lysis be observed within 24 hours of clots from undiluted plasma obtained on the day of the operation. Patient 7 exhibited such a lysis of blood obtained during the first postoperative day.

The data of recalcification time and prothrombin consumption given in Table II indicate there is a more complete clotting at 28° C. than there is at 37° C. In order to establish whether or not this more complete clotting during

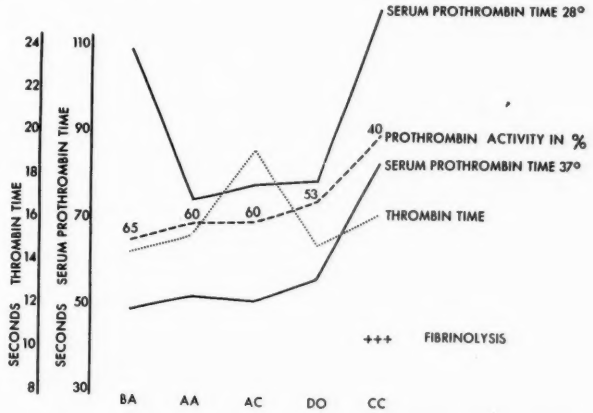

Fig. 1.—Studies of the clotting mechanism in a 24-year-old woman during suture closure of an atrial septal defect under hypothermia. Serum prothrombin time (sec.) specimen, incubated at 37° C. and 28° C.; thrombin time (sec.) at 37° C.; prothrombin activity (%) at 37° C.; and fibrinolysis tendency of recalcified plasma specimens, obtained from blood withdrawn at intervals during the procedure. BA=before anesthesia; AA=after anesthesia; AC=after cooling; DO=during operation; and CC=chest closed.

hypothermia is a real one or only an apparent one, a series of two additional test tube experiments were performed. Thrombin generation tests were performed by a method of our own, as yet unpublished, using recalcified citrated plasma at 37° C. and 28° C., respectively. At intervals, aliquots were taken from the plasma, which was in the process of clotting, and added at 37° C. to a fibrinogen solution which was clotted by it. The clotting speed of the fibrinogen solution is dependent upon the amount of thrombin generated in the clotting plasma, the thrombin amount being related to the amount of thromboplastin formed. Fig. 3 illustrates the thrombin generation in the clotting mixture which was kept at 28° C. proceeds faster and yields more thrombin than in the corresponding mixture incubated at 37° C. This becomes particularly evident if a comparison is made of the clotting times of the fibrinogen solution to which an aliquot of plasma in the process of clotting was added after 12 and 16 minutes of incubation, respectively.

It might be concluded that the increased coagulability at the lower temperature is demonstrable only in citrated plasma, which was used throughout the study, and absent in genuine whole blood. To clarify this point, genuine whole blood, obtained from normal individuals with the two-syringe technique, was

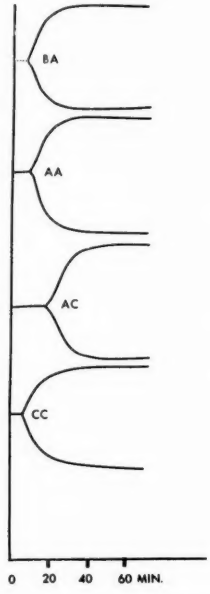

Fig. 2.—Serial coagulograms on Patient No. 5. Fibrin formation begins when the single left side line starts splitting. Note time scale. BA=before anesthesia; AA=after anesthesia; AC=after cooling; and CC=chest closed.

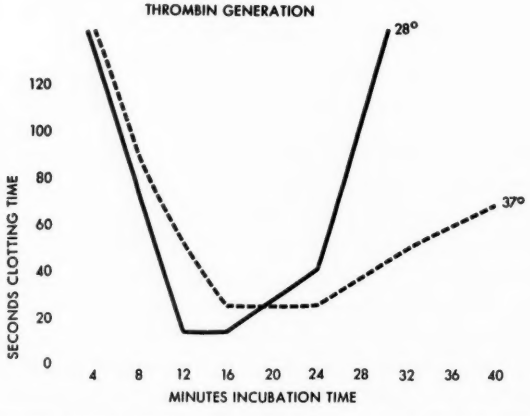

Fig. 3.—Thrombin generation in normal recalcified citrated plasma, incubated at 37° C. and 28° C. Note the formation of more thrombin after a shorter incubation time in the 28° C. specimen.

permitted to clot at 37° C. and at 28° C. in both ordinary and siliconized glass tubes. The clotting time and the prothrombin consumption were measured and reported in Table III. It is evident that whole blood exhibits the same phenomenon as citrated blood. The clotting time at 28° C. is no longer than at 37° C., and the prothrombin consumption is more complete at 28° C. The use of siliconized glassware abolishes this phenomenon. A full account of this and other findings with in vitro clotting studies at various temperatures is now in preparation. A brief preliminary report has been given earlier.[9]

TABLE III

TEMPERATURE (DEGREES C.)	GLASSWARE	CLOTTING TIME (MIN.)	SERUM PROTHROMBIN TIME (SEC.)
37	Ordinary	9	69
37	Silicone	21	22
28	Ordinary	9.3	100
28	Silicone	26	22

DISCUSSION

In a recent paper by Bunker and Goldstein[4] on coagulation in hypothermic patients undergoing major surgical procedures, mainly neuro-surgical in nature, a reduction of prothrombin consumption was reported to occur in some of their patients. It was noted that hypothermia brought about a shortening of the clotting time in some of the patients and the clotting time of the whole blood was not prolonged. The authors discussed the possibility of hypercoagulability or even intravascular clotting at the lower temperature. Using our own in vivo and in vitro studies as a basis, we are inclined to suggest that hypothermia combined with extensive surgical procedures could induce hypercoagulability rather than hypocoagulability, provided there does not develop an increase in thrombin inhibitor. The test tube studies indicate that rough surfaces (i.e., non-siliconized glassware) may enhance this phenomenon. These findings are in contrast to the usual opinion expressed in textbooks that the optimum temperature for blood clotting is 37° C. Bugy,[3] after considering his own data as well as those of others, had concluded the optimum temperature for clotting of human blood is 32° C. He feels this is teleologically justified since, under normal conditions, hemostasis is required at skin temperature and not at that of the interior of the body.

Losner and Volk[12] demonstrated that human blood, to which small amounts of citrate have been added, clotted faster at room temperature than at 37° C. They theorized there was a binding of prothrombin and citrate, a union which became firmer with the increase in the temperature. Our test tube studies with unaltered human blood, showing the same increase in prothrombin consumption at 28° C. as that in citrated blood, seem to exclude any important role of citrate. It is possible that at the lower temperatures some naturally occurring inhibitors of the clotting components are less active. This is well known for substances inhibiting thrombin, and many tests, in which the thrombin formed during the procedures is measured, are done with their final steps at 28° C. to reduce the inactivation of thrombin.

The data presented in Table II demonstrate that cooling of human beings during hypothermic anesthesia does not adversely affect their clotting mechanism as measured by the recalcification time and prothrombin consumption. This is in contrast to the results obtained in dogs by some investigators.[7, 17] This animal responds to cooling with a decrease in platelets which we did not observe in man. Although the clotting and fibrinolytic systems of the dog are basically the same as in human beings, there is a different relationship between the individual factors. A comparative study of clot formation by means of thrombelastograms was made by de Nicola[14] in 18 mammalian species. He found the dog's clot formation began normally, but then progressed very slowly to yield eventually a clot with the poorest firmness of all species studied, man included. It is for these reasons that the results of clotting studies in the dog cannot be correlated with those of man without reservation.

As previously mentioned, recalcification time and prothrombin consumption tests do not indicate impaired clotting during hypothermic heart surgery. However, they do indicate the development of a tendency toward increased clotting during the course of the operation. The explanation for this latter phenomenon is not clear. One may postulate that the release of tissue thromboplastin, particularly from the lungs, may play a role. Interestingly, there appear to be two factors working in opposite directions. The first factor, as seen from Table II, is the prothrombin time. This is usually prolonged in the beginning, and has a consistent tendency to become still longer during the operation in the cooled patient. Patients with congenital heart disease are known to have a prolonged prothrombin time. In cases exhibiting atrial septal defects, the range may be from 15 to 70 per cent,[2] while in patients with tetralogy of Fallot or other cardiac defects, the value may be 10 per cent of normal or lower.[6] It is noted that transfusions with stored blood, which has a more or less pronounced deficiency of its prothrombin complex, certainly do not counteract this abnormality. Ellis, Kleinsasser, and Speer[5] believe that intravascular clotting occurs to some degree in all animals undergoing hypothermia and cardiac surgery. They observed a continuous drop in proconvertin, proaccelerin, and prothrombin during the procedure, and assumed that the reduction of the prothrombin complex and the number of platelets can be explained by the consumption of these factors in intravascular clotting. Bleeding may finally develop due to a lack in the various clotting factors. However, we have not seen a bleeding tendency develop due to a frank deficiency in the prothrombin complex. Normally, the moderate deficiency seen in our patients is not sufficient to influence the over-all clotting mechanism.

In this respect, the second factor, the thrombin inhibitor, is more decisive. The thrombin inhibitor is either identical to heparin combined with its co-factor or is related to this complex. It has been described as being elevated in congenital heart disease, the greatest increase in cyanotic patients.[15] The pre-anesthetic values in our patients (2, 3, and 4) are suggestive of such an increase. The thrombin inhibitor can increase rapidly in response to a variety of stimuli. An

outstanding experimental example of this is the peptone shock which is combined with a considerable increase in capillary permeability.[1] Cooling and rewarming of dogs induced changes of thrombin inhibitor which varied inversely with the temperature.[13] It was observed by de Takats[16] that vasodilatation was associated with an increase in heparin secretion. In 2 of the 3 patients exhibiting an increase in the thrombin titer, bleeding was encountered or was found at autopsy.

It is felt that the increase in thrombin inhibitor and the changes in vascular function associated with it represent the greatest danger in the development of bleeding episodes, and these hemorrhages are not due to the reduction in the concentration of clot promoting factors. If sensitive methods are used, the observed increase in fibrinolytic tendency can frequently be found during chest surgery and does not represent a phenomenon due to hypothermic anesthesia. However, our results do suggest that circulatory occlusion may favor the development of a fibrinolytic tendency.

SUMMARY

The clotting process of human blood as measured by recalcification time and prothrombin consumption is not impaired during hypothermic heart surgery. A tendency for hypercoagulability may develop. Test tube studies show that clotting is as complete at 28° C. as it is at 37° C. Patients with congenital heart disease frequently have prolonged prothrombin times which tend to become longer during hypothermia. Bleeding episodes may be primarily due to an increase in thrombin inhibitor and the changes in vascular function associated with it.

We wish to thank Mrs. Edith von Kaulla for her technical assistance in this study.

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STUDY OF THE VIABLE INTERCOSTAL PEDICLE GRAFT IN TRACHEOBRONCHIAL SURGERY

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INTRODUCTION

THE availability of material within the chest to repair tracheobronchial defects due either to accident trauma or to elective surgery has been demonstrated by a number of investigators.[1-5] In particular, the work of Penton and Brantigan[3] indicated that a viable intercostal pedicle graft could be used successfully. The advantages of the graft are (1) its viability, and (2) the rigid support provided by bone formation. Logically, it might be anticipated that the presence of a blood supply in a graft should ensure success in healing. Specifically, it should encourage the development of a rich capillary network at the critical line of repair. Furthermore, if the vascularity and nerve supply are maintained, the muscle should retain its integrity and reduce the shortening and/or stenosis secondary to fibrosis and atrophy which would develop as blood flow is reduced or abolished. Finally, epithelization should be encouraged, reducing the magnitude of stenosis. The present study was designed to determine the following: (1) the effectiveness of repair with this type of graft; (2) the contribution of the viability of the intercostal vessels; (3) the histologic changes that may occur during healing; and (4) the subsequent results over a prolonged period of time.

MATERIALS AND METHODS

Mongrel dogs were used, anesthetized with Nembutal, and attached to a positive pressure apparatus for respiration on oxygen. Under sterile conditions, the left chest was entered over the fourth rib. The rib was resected and the graft formed from the fourth and fifth intercostal muscle and neurovascular bundles. The graft consisted of intercostal muscle, artery, nerve and vein, periosteum and pleura.[3] In the dog the pleura is extremely thin and, in the early stages of the study, perforations through the pleura occurred frequently. To eliminate this complication, the pleura was imbricated in subsequent animals. The animals were divided into two groups. Group I consisted of animals in which tracheal defects were created and then repaired with the intercostal pedicle graft. The tracheal window was always sufficiently large so as to preclude adequate closure by primary approximation

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and included at least four tracheal rings. In 4 dogs, a tantalum mesh bridge was placed over the defect before application of the graft. Group II consisted of animals in which the left lung was removed, the bronchus being cut flush with the trachea. The defect then was repaired with the intercostal pedicle graft. The graft was placed in such a manner that the serosal surface faced the lumen of the trachea. The closure was effected with interrupted black silk suture using a single layer. Care was taken in the placement of the graft to avoid kinking of the blood vessels and introduction of the suture either through or around a blood vessel. Despite the precaution, interruption of flow did occur in several instances. However, each pedicle graft had two or more neurovascular bundles so that every graft had at least one intact blood and nerve supply.

Following the closure, the intrapulmonary pressure was elevated by increasing the pressure and flow of oxygen to test for leaks. The chest was then closed in anatomic layers and antibiotics were administered for 1 week postoperatively.

RESULTS

Initially, there were a number of deaths due to perforation resulting from undue tension on the suture line. This was avoided in later animals by making a longer graft and allowing for shortening. The animals were observed for periods of 24 hours to 8 months. A total of 34 dogs were considered satisfactory for evaluation. There were 6 deaths. One was due to atelectasis 9 days postoperatively. The others were due to perforations occurring from 36 hours to 4 days postoperatively. Evaluation was based on recovery from surgery, bronchoscopy at regular intervals, and necropsy with microscopic study.

Group I: *Tracheal Defect and Repair.*—Results are indicated in Table I. There were 20 dogs in this group.

Bronchoscopy: Bulging of the graft into the lumen during the first week was observed except in the dogs which had a tantalum bridge. The surface of the graft showed small circumscribed hemorrhagic areas. In the ensuing 3 weeks, the bulging was reduced. A progressively increasing stenosis was observed in most animals after 1½ months. At this time, the surface assumed the gross appearance of the tracheal lining. In the latter months, the trachea was distorted and pulled to the right, in the direction of the base of the graft.

Necropsy: (Fig. 1.) Within 24 hours, the surface was covered with a smooth fibrin lining 1 to 2 mm. thick which could be elevated easily. The graft protruded into the lumen but caused no serious obstruction. The protrusion declined over a period of 1 month at which time healing was practically complete. There was no bulging in dogs with the tantalum bridge.

Granulomas around the sutures were noted occasionally. Tracheal ring regeneration occurred partially and only in those dogs in which stricture was

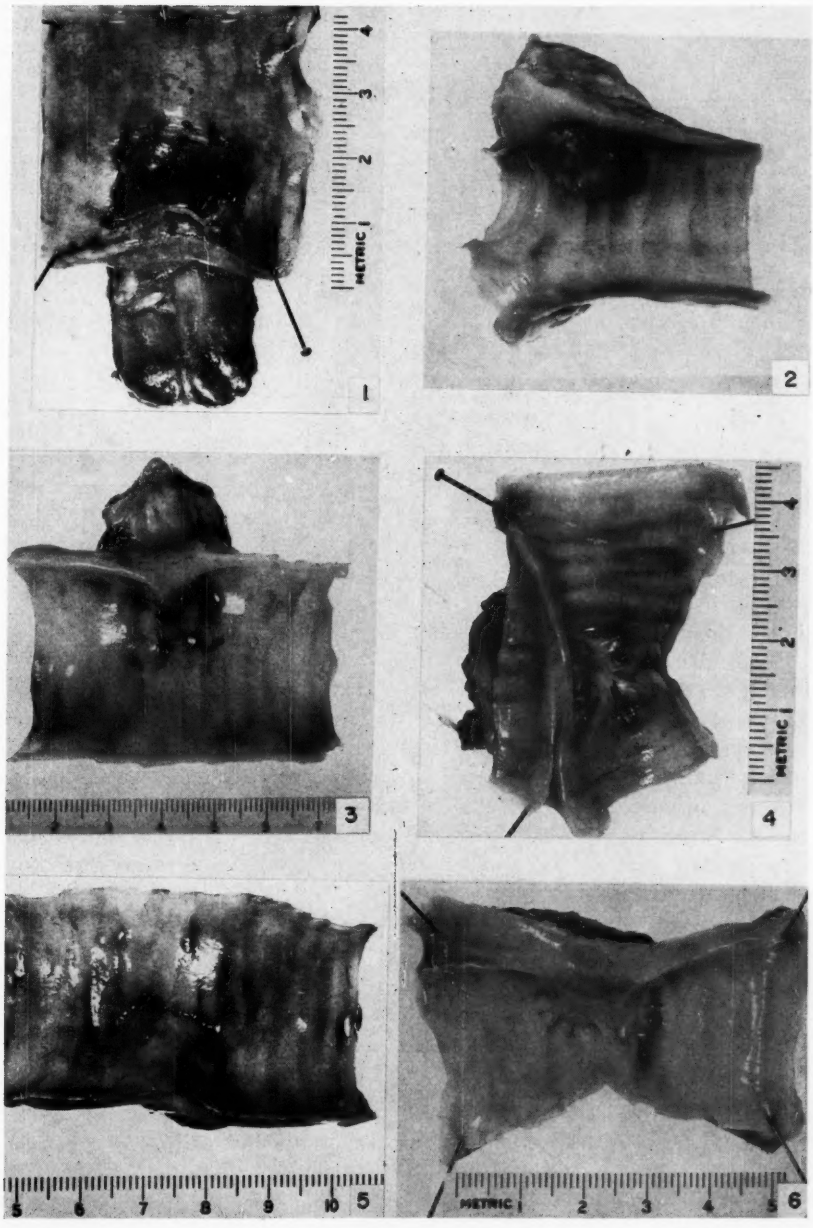

Fig. 1.—Group I. Tracheal Defect. (*1*) Twenty-Four Hours: Note the fibrin deposition. (*2*) Two Weeks: The graft bulges into the lumen. Epithelial islands are apparent. (*3*) One Month: Almost complete epithelial lining. Note silk suture granulomata. (*4*) Three Months: Complete healing with contracture. (*5*) Seven Months: Complete healing with no contracture. (*6*) Seven Months: Contracture. Complete epithelization.

TABLE I. TRACHEAL GRAFT (GROUP I)

NO.	DOG NUMBER	PERIOD OF OBSERVATION	CAUSE OF DEATH	DEGREE OF STENOSIS (%)*	EPITHELIAL‡ GROWTH
1	2083	24 hr.	Sacrifice	0	0
2	589T	36 hr.	Perforation	0	0
3	954	36 hr.	Perforation	0	0
4	803T	48 hr.	Perforation	0	0
5	791	9 days	Atelectasis	20†	0
6	433-A	2 weeks	Sacrifice	8	+
7	286-A	1 mo.	Sacrifice	6	++
8	291-A	1 mo.	Sacrifice	2	++
9	331-A	1 mo.	Sacrifice	27†	+
10	422-A	1.5 mo.	Sacrifice	16	++
11	391-A	2 mo.	Sacrifice	6	+++
12	965	2.5 mo.	Sacrifice	0	++
13	962	3 mo.	Sacrifice	25	+++
14	838T	5 mo.	Sacrifice	30	+++
15	818T	6 mo.	Sacrifice	21	+++
16	775	6.5 mo.	Sacrifice	0	+++
17	762	6.5 mo.	Sacrifice	0	+++
18	759	7 mo.	Sacrifice	0	+++
19	783	7 mo.	Sacrifice	25	+++
20	792	7 mo.	Sacrifice	–	+++

T = Tantalum bridge.

*% Stenosis = $\frac{\text{Normal trachea circumference} - \text{circumference at stricture}}{\text{normal trachea circumference}}$

†No stenosis; bulging of tissue into lumen

‡+ = Beginning. ++ = Partial. +++ = Complete.

minimal or absent. In subsequent months, narrowing of the lumen developed in most animals. The degree of stenosis varied greatly. Of 12 animals followed from 2½ to 7 months, 4 demonstrated no stenosis; and 3 others had stenosis to only a small degree. In those with significant stenosis (over 20 per cent), it was never of such a degree as to embarrass respiration as indicated by subjective observation and by absence of emphysematous changes in the lungs on microscopic study. The tantalum bridge did not modify or reduce the degree of stenosis. Epithelial growth was noted to begin at around 2 weeks, until, within 1 to 2 months, the entire operative site was covered with

TABLE II. BRONCHIAL CLOSURE (GROUP II)

NO.	DOG NUMBER	PERIOD OF OBSERVATION	CAUSE OF DEATH	EPITHELIAL GROWTH*
1	2088	24 hr.	Sacrifice	0
2	741	4 days	Perforation	0
3	732	4 days	Perforation	0
4	801	15 days	Sacrifice	+
5	616	1 mo.	Sacrifice	++
6	819	5.5 mo.	Sacrifice	+++
7	654	7 mo.	Sacrifice	+++
8	668	7 mo.	Sacrifice	+++
9	755	7 mo.	Sacrifice	+++
10	777	7 mo.	Sacrifice	+++
11	681	7.5 mo.	Sacrifice	+++
12	726	8 mo.	Sacrifice	+++
13	719	8 mo.	Sacrifice	+++
14	720	8 mo.	Sacrifice	+++

*+ = Beginning. ++ = Partial. +++ = Complete.

typical tracheal epithelium. This epithelium assumed the usual morphologic characteristics of tall columnar ciliated cells with a distinct basal cuboidal group (Fig. 2).

Group II: *Pneumonectomy and Bronchial Closure.*—The results are indicated in Table II. There were 14 animals in this group of which 2 died 4 days postoperatively due to perforation around the suture lines. The animals which survived from 15 days to 8 months demonstrated excellent healing and complete coverage of the healed area with tracheal epithelium (Fig. 3).

The Intercostal Pedicle Graft.—

Gross: Within a period of 1 month, the graft demonstrated progressive shortening. This shortening resulted in a retraction of the mediastinal contents toward the base of the graft which was lateral and posterior. The graft

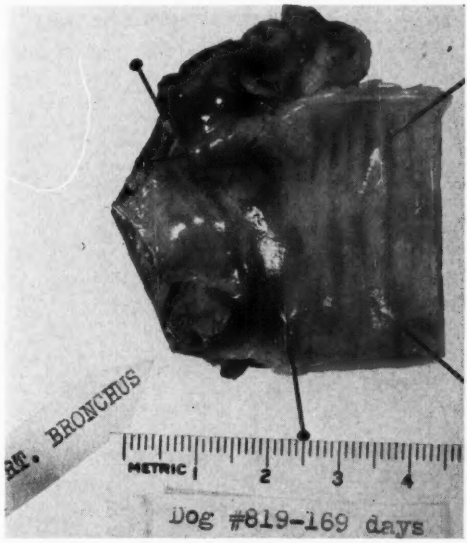

Fig. 2.—Dog from Group II. Open bronchus closed with graft. Well healed with complete epithelial covering.

was firm with considerable bone formation noted frequently. Considerable amount of tissue reaction was demonstrable about the graft investing the trachea and subclavian artery. At no time was the arterial lumen occluded.

Microscopic: The essential microscopic changes in the graft are noted in Table III and Figs. 3 and 4. Within 24 hours, an acute inflammatory reaction occurred at the site of anastomosis. Thrombi of small veins, of some arterioles and, occasionally, an artery were observed. However, in every graft, at least one vascular bundle remained patent. The next interval of 9 days demonstrated recession of the acute inflammatory stage and development of chronic inflammatory reaction with foreign body giant cell activity. At this time, a

rich granulating tissue was growing. During the next week, there was a marked increase in vascularity, including development of vessels to the size of arterioles. The intercostal vessels remained patent. At the end of this time (2 weeks), first signs of epithelial growth at the sides adjacent to the trachea were observed.

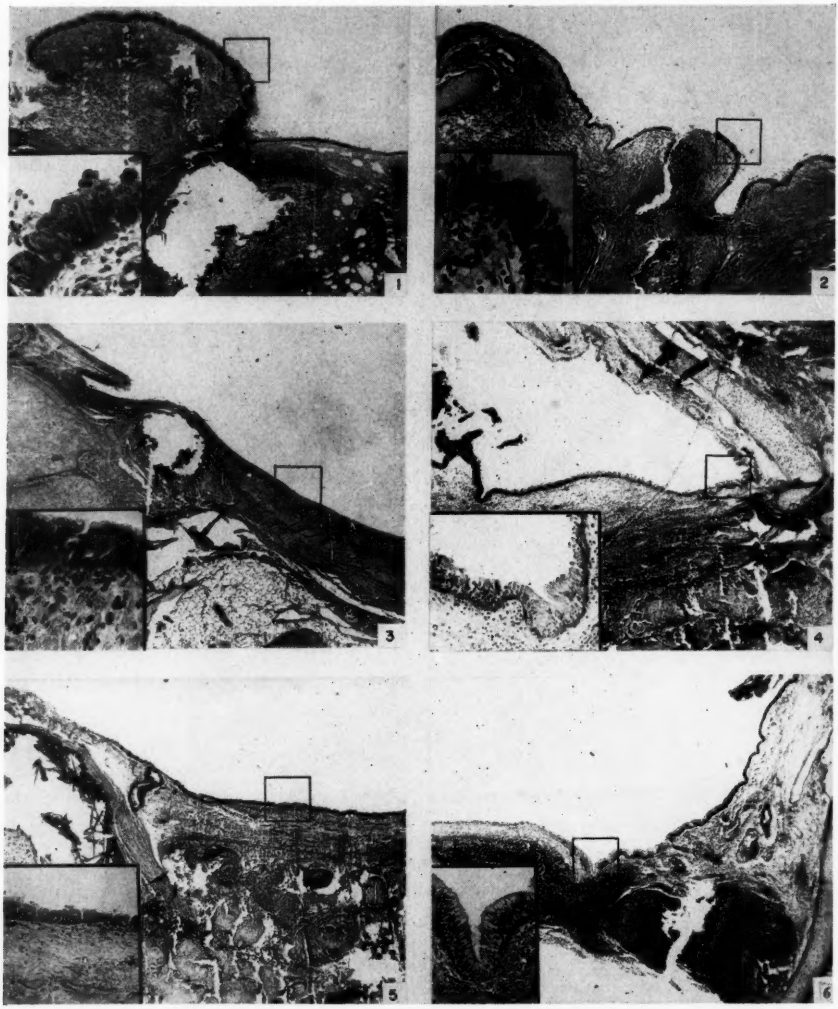

Fig. 3.—Epithelial Growth. Sections are at junction of graft-trachea. Insert is magnified to show epithelial detail. (*1*) Two Weeks: Trachea is at right. The newly growing epithelium is typical tracheal lining and rests on a vascular base. (*2*) One Month: Trachea is at left. Defect was completely covered with epithelium. (*3*) Two Months: Trachea is at right. Muscle in graft is undergoing fibrosis and some atrophy. (*4*) Three Months: Trachea is at left. No tracheal submucosa developed in any animal. (*5*) Seven Months: Trachea is at left. Epithelium rests on a fibrous base. Muscle is fibrosed or atrophic. Note persistent degenerating artery in right mid-field. (*6*) Eight Months: Trachea is at right. Complete atrophy of graft. Chronic inflammatory reaction persists.

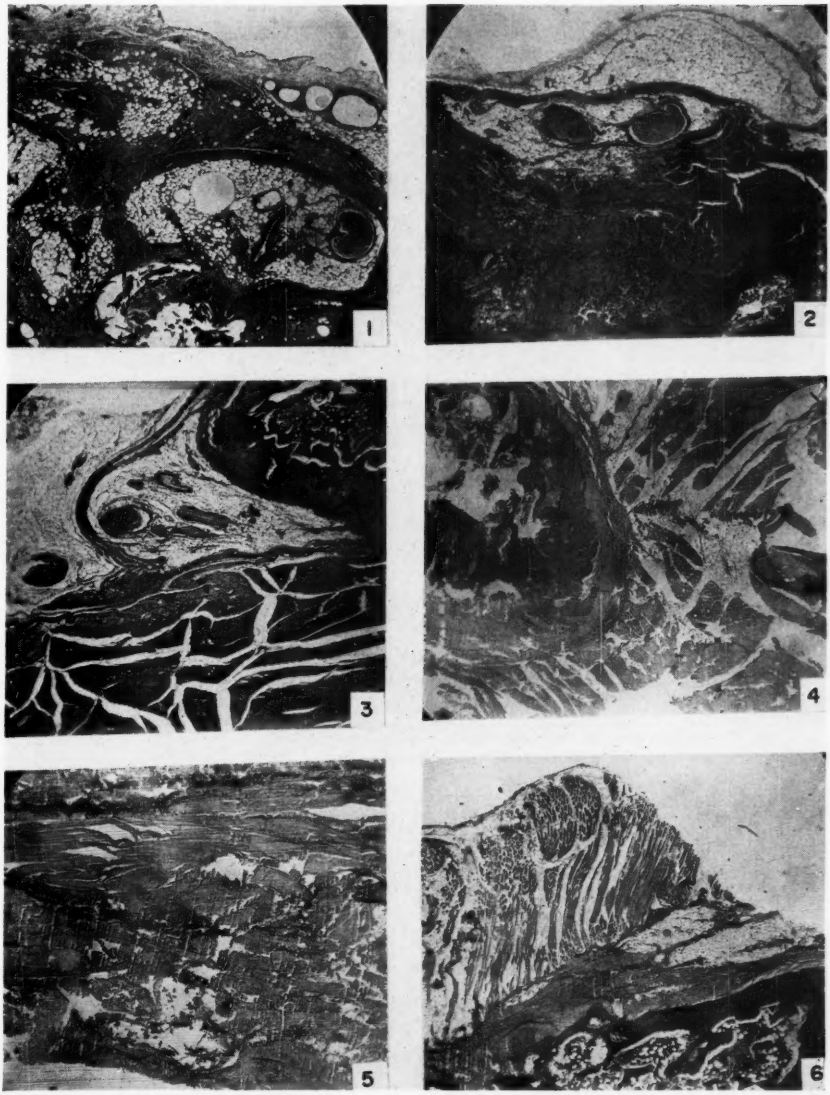

Fig. 4.—The Intercostal Pedicle Graft. (*1*) Two Weeks: The intact neurovascular bundle is at the lower right corner. Note the extensive vascularity, consisting of arterioles and small arteries, in addition to capillaries. (*2*) One Month: The neurovascular bundle is intact. The collateral vascularity is reduced. Chronic inflammatory elements persist and some atrophic and fibrous changes have occurred in the muscle. (*3*) Two Months: Note regenerated rib in upper right corner. The neurovascular bundle is intact. The muscle in this section retains its normal architecture. (*4*) Three Months: Regenerated rib and atrophy of muscle is shown. (*5*) Seven Months: Complete atrophy with replacement of muscle and all vasculature by acellular hyaline material. (*6*) Eight Months: Note presence of some muscle. Graft was almost entirely hyaline and bone.

TABLE III. INTERCOSTAL PEDICLE GRAFT

NO.	DOG NUMBER	PERIOD OF OBSERVATION	MUSCLE*	FIBROSIS†	CALCIUM	BONE
1	2083	24 hr.	N	0	0	0
2	2088	24 hr.	N	0	0	0
3	589	36 hr.	N	0	0	0
4	954	36 hr.	N	0	0	0
5	803	48 hr.	N	0	0	0
6	741	4 days	N	0	0	0
7	732	4 days	N	0	0	0
8	791	9 days	N	Min.	0	0
9	433-A	2 wk.	PA	0	0	0
10	801	15 days	N	Min.	0	0
11	616	1 mo.	N	+	0	0
12	286-A	1 mo.	PA	+	+	+
13	291-A	1 mo.	PA	+	+	+
14	331-A	1 mo.	N	Min.	0	0
15	422-A	1.5 mo.	PA	+	0	Yes
16	391-A	2 mo.	PA	+	0	Yes
17	965	2.5 mo.	PA	++	0	0
18	962	3 mo.	PA	++	0	Yes
19	838	5 mo.	PA	++	Yes	0
20	819	5.5 mo.	PA	++	0	0
21	818	6 mo.	A	+++	Yes	Yes
22	775	6.5 mo.	A	+++	Yes	0
23	762	6.5 mo.	A	+++	Yes	0
24	759	7 mo.	A	+++	Yes	Yes
25	783	7 mo.	A	+++	Yes	Yes
26	792	7 mo.	A	+++	Yes	Yes
27	654	7 mo.	A	+++	Yes	0
28	668	7 mo.	A	+++	Yes	0
29	755	7 mo.	A	+++	Yes	0
30	777	7 mo.	A	+++	Yes	0
31	681	7.5 mo.	A	+++	Yes	0
32	726	8 mo.	A	+++	Yes	Yes
33	719	8 mo.	A	+++	Yes	0
34	720	8 mo.	A	+++	Yes	0

*N = Normal. PA = Partial atrophy. A = Atrophy.
†+ = Moderate. ++ = Subtotal. +++ = Total.

In 1 month, epithelization was almost complete. Chronic inflammatory elements persisted while the granulation tissue and capillary activity receded and were being replaced by fibrous tissue. Collaterals, most likely developing from the intercostal arteries, were abundant. The muscle retained its normal architecture.

Significant degenerative changes began to appear between 1 and 2 months. The inflammatory reaction had subsided considerably, but macrophagic activity was still present. The muscle began to show signs of atrophy in the form of hyaline degeneration. The degree of fibrosis increased. The intercostal neurovascular bundle was still intact. In this period, epithelization was complete. Serious muscular atrophic changes occurred in the next 3 and 4 months despite the persistence of patent intercostal vessels. The capillary network had disappeared, and the collaterals were being replaced gradually by fibrous tissue of hyaline materials. The fifth to the sixth postoperative month marked the period of obliteration of the neurovascular bundle, and, except in a few instances, replacement of the muscle by hyaline material and

contracted, relatively avascular fibrous tissue. In the seventh and eighth months, the graft consisted of masses of hyaline material and contracted fibrous tissue occasionally surrounding bone.

DISCUSSION

The intercostal pedicle graft is easily mobilized as sufficient tissue is available for the repair of the defects created in this study. The procedure was relatively simple to complete, and the results quite satisfactory. With regard to viability, it appears that the blood flow through the intercostal vessels contributed to the early phases of healing and to delaying the atrophic changes in the muscle. The presence of a very rich granulation tissue ensured adequate vascularity for healing. Further, as opposed to free muscle graft,[9] large caliber collaterals also developed. This indicated a greater vascularity than could be achieved with a free graft and likely contributed to the delay in atrophic changes of the muscle. Peer has documented that free muscle becomes completely atrophic within 1 month.[9] He observed that muscle undergoes atrophy when it is immobilized and not in use. This disuse atrophy developed in every dog in the present study, though at a much later period (6 months). At approximately the same time, the neurovascular bundles disappeared.

The deposition of calcium and the formation of bone developed as demonstrated elsewhere.[3] The fairly early appearance of tracheal epithelium and eventual complete coverage has been consistently observed.[3, 6, 7, 8, 10]

Pleura alone or fat has been used with success in the closure of bronchial stumps.[1, 2, 4, 5] The results of these studies indicate that a viable graft is not necessarily required to assure adequate closure of a bronchial stump or tracheal defect.

The success in healing of tracheobronchial defects, as established in this study, was due to the classic mechanisms of healing involving aseptic, careful surgery, the deposition of granulation tissue, and the eventual union by fibrosis. The viability, while contributory, does not eliminate the eventual atrophic changes, nor does it accelerate epithelization nor prevent stenosis. If there is any question of delay of union or healing because of suspected ischemia or infection, the advantage of a higher degree of vascularity may indicate the use of a viable graft.

CONCLUSIONS

1. A viable intercostal pedicle graft consisting of intercostal muscle, the neurovascular bundle, periosteum and pleura was employed to repair tracheobronchial defects.

2. The repairs were eminently successful and, although significant degrees of stenosis developed in the trachea, there was no evidence of pulmonary emphysema through the period of 7 months in which these animals were followed.

3. While the viability of the graft may have contributed to the successful healing, union was due to the fundamental classic mechanisms of tissue healing. The viability did not prevent tracheal contracture or atrophy of the graft.

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